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INTERSTITIAL NEURITIS AND THE PRESSURE THEORY OF PAIN: A CRITICAL REVIEW AND A REFLEX HYPOTHESIS.[1]

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NEURALGIA AND NEURITIS.

INTERSTITIAL NEURITIS, or fibrositis of the perineurium, is characterized by four main signs or symptoms (Harris, 1926; Walshe, 1943): (*a*) radiating pain; (*b*) tenderness in the vicinity of a nerve trunk; (*c*) paræsthesiæ or objective sensory loss; (*d*) muscular wasting or paresis, or loss of deep reflexes. If the distribution of the pain and tenderness, however, is such as to suggest the involvement of a nerve trunk, the diagnosis may be made in the absence of motor and sensory impairment, which, it is generally agreed, are encountered only in a minority of cases.

Little is known of the pathology of neuritis. Symonds (1943) contends that it is built upon guesswork, for the neurologist rarely sees the nerve which is thought to be affected. Most authorities agree, however, that the interstitial tissues of the nerve are inflamed, this causing pain which is at first local, but which later spreads more widely through compression of the fibres of the nerve trunk.

Neuralgia.

Harris defines neuralgia as a radiating pain which spreads far more widely than the territory of the diseased nerve filament which initiates it. Sometimes it embraces the field of a peripheral nerve; a good example is the behaviour of a carious tooth, in which the pain sometimes spreads to involve one or more divisions of the trigeminal nerve. "Violent and widespread neuralgia", wrote Harris, "may therefore be a sensory reflex from an acute neuritis of a few nerve filaments. . . . One is tempted to assert that all neuralgic attacks are reflexes from an irritative focus."

Most authorities find difficulty in differentiating neuralgia from neuritis. Frazier, Lewy and Rowe (1937) prefer to speak of neuralgia only when no organic lesion of any kind can be found. Paget's definition of neuralgia (1876) agrees closely with that of Harris; and it seems to be generally agreed that, while in neuritis a considerable area is involved through compression of its nerve trunk, in neuralgia the pain spreads over a large area indirectly through reflexes generated in a minute sensory fibril.

Recent advances in the study of pain have supported the prediction of Harris that many neuralgias would prove to be due to reflexes from irritative foci. The independent efforts of several groups of workers have dovetailed in a striking fashion to give a picture of the mechanism by which pain spreads. If pain spreads from a small focus to involve the field of a major nerve, the impulses would first have to travel in a central direction, and, at the branchings of the axones, would reverse their direction and travel antidromically to the periphery. And the work of Sir Thomas Lewis (1942) suggests that this may happen.

The Spread of Cutaneous Hyperalgesia.

Lewis demonstrated that the spread of hyperalgesia from a point of injury in the skin was brought about through a neural mechanism. Faradization of a digital branch of the ulnar or the median nerve, moreover, was followed by hyperalgesia throughout its distribution in the hand. The manner of spread here closely resembles that which is postulated for the spread of neuralgic pain. Lewis thought that a hitherto undescribed system of nerves was involved;

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[2] Working with a grant from the National Health and Medical Research Council.

but Woollard, Weddell and Harpman (1940) held that the cutaneous pain nerves possessed all the anatomical attributes necessary for such a system.

Again, by provoking painful impulses from deeper tissues, Lewis produced hyperalgesia in the distribution of a sensory nerve. Thus, after faradization of the wall of the antrum, hyperalgesia was observed throughout the field of the maxillary division of the trigeminal nerve.

The Deep Pain System.

Lewis, assisted by Kellgren, studied the responses given by many tissues to needle puncture. Many fibrous and supporting tissues were found to give pain on puncture; and the sensation differed strikingly from cutaneous pain. Muscle tissue, which is insensitive to puncture, gives pain when a small quantity of hypertonic saline solution is injected into it. Deep pain is aching in character if less intense, and "neuralgic" if more intense. It radiates diffusely over wide areas, and closely resembles visceral pain. It tends to follow regular segmental patterns, which, however, do not correspond with the segments of cutaneous sensory distribution. Inman and Saunders (1944) confirmed all of these observations.

It was observed further that cutaneous hyperalgesia, muscular rigidity and deep tenderness often accompanied referred somatic pain, even as they are known to accompany referred visceral pain. The deep tenderness develops with the pain and passes off when the pain abates. Cutaneous hyperalgesia, however, is seen less frequently, and its distribution does not correspond with that of the deep tenderness. Lewis drew the conclusion that the deep pain system, which comprises both visceral and somatic pain, is separate, both anatomically and functionally, from the cutaneous pain system.

Since cutaneous hyperalgesia spreads by nervous reflexes through a superficial system of nerves, it was argued that the spread of deep tenderness was due to a similar process in the deeper tissues. It will be noted that the radiation of pain and tenderness from a deep lesion presents the features of neuralgia as defined by Harris.

Fibrositis.

Though the pathology of fibrositis is obscure, there is little doubt that the somatic pains occur to which the name is commonly applied, and its use is justified for the sake of convenience.

Following the work of Kellgren (1938), local anæsthesia has become a standard means of investigation and treatment of fibrositis. Widely radiating pain, with diffuse tenderness, can be relieved by anæsthetizing a localized tender spot in the deep tissues. When cutaneous hyperalgesia occurs, its distribution does not correspond with that of the deep tenderness. Fibrositis, it will be seen, corresponds closely with Harris's conception of neuralgia as a reflex sensory phenomenon.

Painful Post-Traumatic States.

In America some attention has been devoted to those painful conditions which occasionally follow injuries. The pain, dysæsthesiæ, œdema and tissue atrophy are thought to be reflex effects, the impulses originating in the damaged tissues (Livingston, 1943; Miller and de Takats, 1942). The writers frequently observed patches of hyperalgesia or hypoæsthesia which did not correspond with the distribution of nerve trunks. These, too, they thought to be reflex effects.

Thus the reflex hypothesis is applicable to yet another variety of spontaneous somatic pain. Such an hypothesis, however, is futile if it does not correspond with the known anatomical and physiological qualities of peripheral sensory nerves. Recent advances in microscopic anatomy and in neurophysiology, however, have established (*a*) that such a system exists and (*b*) that it possesses the requisite functions.

The Pain Plexus.—Lewis presumed the existence of a special "nocifensor" system of nerves which mediates the spread of hyperalgesia. The majority of observers, however, now agree with Woollard that the pain plexus has all the requisite qualities (Walshe, 1942). In addition, a plexus resembling that of the skin is found in those deep tissues which are sensitive to puncture with a needle (Weddell and Harpman, 1940).

Bidirectional Function of Sensory Nerves.—The antidromic propagation of impulses suggests that nerves normally may be capable of propagating impulses in either direction. Toennies (1938) and Barron (1940) have been able to demonstrate an efferent outflow through adjacent posterior roots in response to the stimulation of a sensory nerve. Adrian, Cattell and Hoagland (1931) showed that stimulation of the touch receptors in the skin of the cat and the frog had the effect of reducing the activity of adjacent receptors. Hyperalgesia normally results from the antidromic stimulation of a sensory nerve (Lewis); and Thompson (1931) was able to produce anæsthesia in man by faradization of cutaneous nerves.

Interstitial Neuritis.

The use of the term interstitial neuritis implies the existence of a lesion whose existence never has been confirmed. When the sheath of a nerve trunk becomes involved in a fibrositic process, says Harris, the pain spreads more widely because of two additional features: (*a*) involvement of the *nervi nervorum*; (*b*) pressure upon the axis cylinders.

But, when discussing neuralgia, we have seen that pain from many deep lesions radiates widely, probably through the medium of nervous reflexes. Puncture of an arterial coat, for instance, causes a widespread neuralgic pain, often with such reflex effects as faintness (Bazett and McGlone, 1928). The pain plexus is found in the adventitious coat of an artery; and a similar plexus is found in the epineurium, which responds to injury by giving off grouped discharges resembling those from a sense organ (Adrian, 1930). To explain the pain of neuritis, therefore, it is not necessary to invoke pressure on the axis cylinders.

Neuritis is distinguished from neuralgia by the supposed existence of a lesion of the nerve trunk, which causes local tenderness, or pain on stretching of the nerve. It is only in a minority of cases that the lesion causes motor or sensory loss; thus we are asked to assume that pressure upon the fibres causes pain long before it interferes with conduction. In other words, the orthodox conception of neuritis depends upon the view that pain is the earliest effect of pressure on nerve trunks. This view in its turn is the offspring of the widely held opinion which relates pain of every kind to pressure on nerves—the pressure theory of pain.

THE PRESSURE THEORY OF PAIN.

There is a general assumption in medicine that pain is the result of pressure, that any spontaneous pain stands explained if the existence of pressure or tension upon nerves can be assumed. The result is that, in their attempts to explain a pain, authors often make more use of ingenuity in speculation than of scientific inquiry, and any explanation will do if it can only be shown that this mechanical factor is operating.

But there exists no proof that pressure is an important factor in the genesis of pain. It is well known that pressure upon a nerve may result in painless paralysis (for example, crutch palsy). Medium pressure on the ulnar nerve interrupts conduction without pain; the observer can confirm this. This was known to A. Waller (1862) and to Head (1896), who wrote: "pressure on nerve trunks causes numbness and tingling, but not superficial tenderness". Firm compression is painful, but this is not necessarily due to pressure on axis cylinders, for a similar response can be obtained from certain spots in muscle, and lighter pressure upon the testis or the eyeball results in pain of greater intensity.

Regarding the qualities of the fibres which convey pain, the classical work initiated by Adrian and Gasser has not yet come to fruition, and it cannot be asserted that there is any general correlation between the diameter of a fibre and its function (Gasser, 1943). In response to external pressure, motor and other sensory fibres are blocked before

the pain fibres (Lewis; Gasser; Tower, 1943; Denny-Brown and Brenner, 1944).

Clinically, pain is not a prominent feature either of injuries to nerves (Seddon, 1943; Sunderland, 1946) or of tumours of nerves, malignant (Stout, 1935) or benign (Mason, 1937; Erlich and Martin, 1943). Bishop (1944) found it difficult to stimulate the pain fibres by faradization of a small cutaneous nerve; though other feelings were evoked, prick sensation could not be elicited in its distribution.

But it may be argued that these examples are not analogous to neuritis, in which poisoning of fibres may be an additional factor. Many instances can be cited of paralysis which was preceded for a few days by severe pain (Burnard and Fox, 1942; Richardson, 1942; Spillane, 1943). It has been known for many years, however, that nerve fibres are almost unfatigueable, and are extremely resistant to ischæmia and to toxic influences of all kinds (A. D. Waller, 1897; Sherrington, 1906; Heinbecker, 1929; Bentley and Schlapp, 1943).

The pain of interstitial neuritis, therefore, is not elicited by damage to axis cylinders. The painful stimulus probably is not a mechanical one, for no pathological state is capable of developing the necessary degree of pressure.

Experimental Observations on Painful Pressure.

As a general rule, mechanical pressure, when intensified, increases to pain. That this pressure-pain sensation is not located in the skin can easily be shown. Heavy pressure or a blow upon the tibia through anæsthetized skin is painful; pressure through normally sensitive skin upon anæsthetized periosteum is painless. Similarly, pressure upon muscle through anæsthetized skin is painful. Compression of a fold of loose skin between finger and thumb is not painful if the skin is not sharply angulated and pinched (Waterston, 1931).

But pressure sense and pain sense are not identical, for periodontal membrane is extremely sensitive to pressure but not to pain. Tooth pulp, however, is sensitive to the lightest touch; but in a pulpless (that is, painless) tooth, the pressure sense is not diminished (Lewinsky and Stewart, 1937). In the sole of the foot, pulps of fingers and many other regions, great pressures can be borne without pain. Perhaps deep pressure pain is the same as the deep pain response to needle puncture; it may be that its receptor is the pain plexus in the deep fascia.

Most of the recorded experiments dealing with pressure pain were intended to locate the pathways of the impulses rather than the situation of the receptors (Head and Sherren, 1905; Sherrington, 1909; Stopford, 1923; Duthie, 1926). Simple tests can be designed, however, for the latter purpose.

Muscle.—A needle is passed obliquely to the surface of the muscle, and two cubic centimetres of procaine solution are rapidly injected. Within five seconds pressure pain is completely abolished over an area an inch in diameter. Similarly, some more sensitive spots, usually near the musculo-tendinous junctions, can be rendered painless to pressure by anæsthetizing the surface of the muscle.

Tendon.—It can be shown also that "tendon sense" is located in the epitenal tissues. The substance of the tendon is insensitive to pressure, even as it is to puncture (Weddell and Harpman).

Deep Hyperalgesia.—In an induced state of deep tenderness, is the sensitive layer (epimysium *et cetera*) abnormally tender, or have other tissues (for example, muscle) become sensitive which are normally insensitive to pressure?

In one experiment performed by the writer upon himself, a spot of heightened sensitivity in the left gastrocnemius muscle was anæsthetized. Afterwards the whole of the calf became extremely tender, and the tenderness extended up to the posterior surface of the thigh. Cutaneous sensation was normal. It was now possible to abolish pressure pain over a small area by anæsthetizing the surface of the muscle. Deep hyperalgesia, therefore, consists of a state of heightened sensibility of the pressure-pain receptors.

A Biological Theory of Pain.

Such a biological phenomenon as pain cannot be explained adequately on mechanical grounds. The function of the pain sense is to herald impending damage (Trotter and Davies, 1909), and the deep receptors respond to pressure when it becomes harmful. But mechanical stimuli are not essential; thus pain can be aroused from muscle by contraction under ischæmic conditions, or by the injection into it of small quantities of hypertonic saline solution, or spontaneously by a fibrositic lesion. The deep pain of a muscular lesion is not at all like cutaneous pain, and it is distributed in different patterns. Dejerine (1926) called these segments of deep sensation "sclerotomes" to distinguish them from the dermatomes. Head, Rivers and Sherren (1905) observed that deep sensation was separate from cutaneous sensation, though they did not clearly distinguish deep pain from cutaneous pain; both were included by them in the protopathic system. Section of a cutaneous nerve does not abolish the pain felt on pressure in the area; rather it heightens it (Sharpey-Schafer, 1927; Head *et alii*; Seddon).

The spontaneous pain of many diseases is accompanied by tenderness of the deeper structures rather than by cutaneous hyperalgesia; and much evidence suggests that it is a product of the deep pain system. The vast bulk of research upon pain, however, has dealt with the cutaneous receptors, and it is possible that many of the conclusions drawn therefrom are not applicable to the pain of disease.

The Deep Pain System and the Pain of Disease.

Leriche (1939) asserted that the pain of disease differed vastly from cutaneous pain. Lewis produced evidence suggesting that deep somatic pain is served by the same nervous mechanism as is visceral pain. This mechanism is separate from the cutaneous pain system of nerves; it has freer connexions and thus radiates more widely. In attempts to understand it, the significance of the deep tenderness which accompanies it should not be forgotten.

The deep tenderness which accompanies an artificial muscular lesion is the result of stimuli, originating in an insensitive structure, which by nervous reflexes cause a change in the state of receptors in the adjacent epimysium. The appearance of tenderness indicates that a physical change has taken place. The spread of hyperalgesia, said Lewis, can be likened to a process of secretion; a chemical substance is liberated which sensitizes pain receptors. Lewis does not exclude nervous or other mechanisms in the liberation of "factor P" from ischæmic muscle, and the analogy with the effects of muscular lesions is a close one; possibly the same factor is involved. "Factor P" must be very unstable, because the tenderness disappears as soon as the lesion is anæsthetized.

Referred pain usually is accompanied by tenderness, and it is not improbable that the pain, like the tenderness, is a truly reflex phenomenon, in which pain receptors over a large area are sensitized by antidromic impulses. The radiating, spontaneous pain of disease, perhaps, is the result of the induced activity of a large number of pain receptors.

Pain receptors occur in many tissues inaccessible to palpation. Sometimes a pain which radiates widely and deeply is not accompanied by tenderness, perhaps because of the inaccessibility of the receptors. When an area is diffusely tender, all the structures which possess a pain plexus (arterial coats, epineurium *et cetera*) become tender. When a nerve trunk is tender, however, its contribution to the general tenderness may not be important.

Fibrositis and Local Analgesia.

The commonest example of spontaneous pain is fibrositis, a tissue reaction whose pathology is not well defined. Pain may occur in any region, due to a well-defined lesion which is responsible for diffuse pain and tenderness. The value of local analgesia in its diagnosis and treatment has been confirmed by a very large number of observers. The injection of procaine into the lesion may be followed by the disappearance of pain and tenderness some distance away; thus tenderness in the hand or wrist may vanish

after treatment of a lesion in the *extensor digitorum communis* near its origin from the lateral epicondyle.

Fibrositis in every region displays the same characters, which have been described in a number of papers (Kelly, 1942, 1944). Some of these examples may be considered briefly.

Headache.

Pains in the head due to various causes may be associated with lesions in the suboccipital muscles (see also Jones and Brown, 1944). In some instances of distressing neuralgic pain dramatic cures have been obtained.

Pleurodynia and Abdominal Pain.

Intercostal neuralgia may be attributable to a lesion in an intercostal space, while obscure abdominal pains may be explained upon similar lines. Pains in the trunk, however, are not commonly called neuritis; this term usually is reserved for pains in the limbs, which often radiate through the length of the extremity and are associated with deep tenderness. The course of the main nerve trunks is such that one of them may traverse a tender area, and a natural deduction may be that the nerve itself is tender.

"Brachial Neuritis."

Fibrositis is the commonest cause of pain which radiates from the shoulder to the hand (Gowers, 1904; Stockman, 1940). It is usual to look for mechanical causes for such pain, and, though the fashions in interpretation have changed, pressure upon nerve roots or the brachial plexus has been the basis of them all. Cervical rib, "pseudocervical rib", scalenus syndrome, arthritis of cervical spinal joints, and cervical intervertebral disks—each in turn has enjoyed its vogue. There is little doubt that sensory and motor defects can result from the cervical rib syndrome, but circulatory impairment is more common (Walshe, Jackson and Wyburn-Mason, 1944). But the common syndrome of pain in the arm usually can be explained upon the simpler basis of a muscular lesion. Like somatic pain elsewhere, it tends to assume constantly repeated patterns, and four distinct syndromes are common. More than one lesion may be responsible, and local analgesia is of assistance in resolving the syndrome into its component parts. Later experience has confirmed the earlier impressions of the writer (1942, 1944): (i) When pain radiates from the shoulder to the hand, a lesion in the scapular muscles (commonly in the supraspinous region) coexists with another in the extensor muscles of the forearm. (ii) Less commonly, pain radiates to the hand from a single lesion, situated in the paravertebral muscles in the cervico-dorsal region. (iii) When the supraspinous lesion occurs alone, the picture usually does not suggest neuritis, the pain radiating only to the middle of the humerus. (iv) A single lesion in the extensor mass just below the epicondyle is capable of causing pain which radiates from the lower third of the humerus to the fingers, or which may be localized to any part of this wide area.

One hundred and thirty-eight cases of fibrositic pain in the upper limb have been investigated. In rather more than a third lasting abatement was obtained; in most of the remainder relief lasted for the duration of the analgesia only. Forty-two of these cases could have been diagnosed as interstitial neuritis, because of the pain radiating to the fingers and of the situation of the tenderness; motor or sensory impairment, however, occurred in only fourteen of these.

Of the forty-two cases, seventeen belonged to category (i), with lesions in the shoulder girdle and the forearm; lasting relief of one or both lesions was secured in eleven. Eight belonged to category (ii), and three patients were relieved. Seventeen were of category (iv); ten patients were relieved by local analgesia.

Where muscular paresis or cutaneous hypoæsthesia was found, these signs did not show improvement for some time. In several such cases, however, treatment of muscular lesions completely relieved the neuralgic pain which was the chief complaint. On only one occasion was a nerve blocked by the anæsthesia; this seems to suggest that the deep tenderness of "neuritis" is not commonly related to a nerve trunk.

In treatment of painful shoulders, Travell, Rinzler and Hermann (1942) obtained results which corresponded with those of the present writer. Pain in the forearm due to muscular lesions near the elbow has been described by Good (1942). Judovich (1945) recently described a case of "scalenus syndrome" in which the pain was divided into a shoulder component and a forearm component. The former was relieved by anæsthetizing a lesion in the *scalenus anterior*, and the latter by treating a lesion in the extensor mass.

"Sciatica."

We have seen that pain in the arm tends to spread in fairly constant patterns, frequently with some limitation of shoulder movements. In the leg the pattern is still more constant, taking the familiar form which we know as sciatica.

In thirty-four of forty-six cases the symptoms were the usual three: (i) pain radiating from the buttock to the lower part of the calf or the heel; (ii) tender areas in the buttock and posterior part of the thigh, and sometimes the calf; (iii) pain on flexing the hip with the knee straight. In the remaining twelve, sensory or motor impairment was seen as well.

As in the case of brachial neuritis, the infiltration of deep lesions with procaine usually abolishes the pain, tenderness and limitation of movement (Steindler, 1940; Kellgren, 1941; Le Vay, 1944); but permanent benefit was secured in only nine of the writer's cases. The relief by treatment of muscular lesions suggests that the tenderness is not of the nerve, but of the deep fascia related to the gluteal and hamstring muscles; and the limitation of straight leg raising is due to stretching of the irritable hamstring muscles rather than of the nerve (Purdon Martin, 1942; Le Vay). Motor and sensory signs usually are limited to the distribution of the fifth lumbar segment; wasting of the calf muscles and dysæsthesia above the lateral malleolus are the commonest signs.

A great deal has been written of late on sciatica and the intervertebral disk; it is not opportune therefore to discuss at length the question of the nature of sciatic pain. It should be reemphasized, however, that there is little positive evidence that root pressure commonly produces pain; and that pains of a similar type in other regions can be explained upon a different pathological basis.

The Symptomatology of Interstitial Neuritis.

Little is known of the pathology of interstitial neuritis, because of the impossibility of securing specimens for examination. A few autopsies have been reported, in which the sciatic nerve showed no change; in that of Denny-Brown (1933) the changes were not significant. Purdon Martin (1933) found the pain hard to explain; it could not, he said, be due to pressure alone, and perhaps there was in addition an altered state of the *nervi nervorum*.

Usually, in fact, there is no direct evidence of a lesion of a nerve trunk. Tenderness cannot be attributed to the nerve when it lies deep to structures which themselves are tender. When pain occurs on a movement which is thought to stretch the nerve, the same manipulation puts strain on muscles which may be irritable. In the majority of cases in which interstitial neuritis is diagnosed, the signs can be wholly explained upon the known nature of the fibrositic lesion. The existence of a nerve lesion has only been assumed, from the nature of the rheumatic process and from the fact that the more severe cases may present signs of a trunk lesion.

Differences between Interstitial Neuritis and Traumatic Lesions of a Nerve.

When muscular paresis and cutaneous sensory loss occur, a close resemblance is presented to mechanical block of a nerve trunk. But the general picture of a fully developed neuritis may differ in four important details from that of injury to a nerve: (*a*) the pathology of the lesion, (*b*) the incidence of pain and tenderness, (*c*) the distribution of the manifestations, (*d*) the prognosis.

Pathology of the Lesion.

In interstitial neuritis it is generally presumed that an inflammatory lesion of the sheath presses upon axis cylinders. It is characteristic of rheumatic inflammation that the reaction produces agencies which damage the vitality of parenchymatous tissues. In some cases of neuritis the tender nerve can be felt directly, and it would be futile to deny the probability that it is inflamed. But the purpose of this essay is to advance the opinion that the accepted explanation is inadequate in many cases.

In a classic study of traumatic lesions in continuity, Seddon was able to draw the conclusion that good recovery could be expected if the neural tubes were intact. When the tubes had been disorganized, neuromata or thickenings of the nerve always resulted, and the quality of recovery was poor. The type of lesion in interstitial neuritis should correspond with the less severe lesions in continuity, in which the prognosis is good. In neuritis complete recovery fails to occur in an appreciable proportion of cases; but in spite of this, there never has been recorded a case in which a thickening of the nerve indicated disorganization of the nerve trunk.

The negative evidence provided by pathology, therefore, is of value in indicating a fundamental difference between interstitial neuritis and a proven nerve lesion.

Pain and Deep Tenderness.

Authorities have been cited who agree that pain is not a common feature of injury to a nerve. Causalgia was first reported by Denmark (1813), but it is an uncommon sequel. In the gradual or rapid destruction of a nerve (by pressure, by tension or by tumour growth) pain and tenderness are not prominent, while they are outstanding features of neuritis. In Cases XI and XII the lateral popliteal nerve was interrupted by compression, and Case XIII was one of tourniquet paralysis of the radial nerve. All three conditions were painless. In Case XIV, on the other hand, the picture was one of post-malarial neuritis of both divisions of the sciatic nerve, and pain preceded the onset of complete motor and sensory paralysis below the knee. The muscles supplied by the anterior tibial nerve failed to recover. In Case XV a similar picture came on spontaneously, preceded by severe pain; but recovery after six months was complete. In Case XVI pain followed a slight blow to the wrist; complete dorsal interosseous paralysis ensued, followed by recovery.

In Case XVIII the circumflex nerve was injured when the shoulder was dislocated, and the deltoid paralysis was painless. In Case XX, on the other hand, deltoid paresis came on after severe pain of two weeks' duration; the pain was cured by anæsthetizing a small lesion in the fibres of origin of the deltoid.

Many more such cases could be cited, indicating that pain is a special feature of interstitial neuritis which clearly differentiates it from a simple lesion of a nerve trunk.

Anatomical Distribution of Manifestations.

When a nerve trunk is interrupted by mechanical means, the muscles affected and the extent of the sensory loss are strictly related to the distribution of the nerve. In interstitial neuritis, on the other hand, an overflow into the fields of other nerves is not uncommon. In Case XXI a lesion involving the circumflex nerve overflowed to affect cutaneous branches of the posterior cord of the plexus. In Case XVII, which presented similar features, a posterior cord lesion could not be diagnosed, because of the added palsy of the suprascapular nerve, a branch of the upper trunk. Here the paralysis occurred in the left shoulder five weeks after an injury to the right shoulder, which had been followed by pain on that side for a few weeks.

In Case XVI a blow on the wrist was followed by dorsal interosseous paralysis; and in Case XXXVI a mild crushing injury to the right hand was followed by pain in the hand, and later by anæsthesia throughout the ulnar distribution, which extended up to the lower third of the forearm. Case XXVIII was one of musculo-cutaneous neuritis, in which paralysis of the biceps was complete, but that of the brachialis incomplete. The senory loss, however, embraced the whole of the forearm and hand, indicating that the disorder had spread from the musculo-cutaneous to involve other nerves in an irregular fashion. Similar cases have been reported in British and Dominion armies (Burnard and Fox; Spillane). The pathological process seems to be able to overflow into the distribution of other nerves, even as, on a lesser scale, neuralgia is defined as an overflow from small pain fibres to larger nerve trunks.

The tendency of neuritis to spread has not been satisfactorily explained. For many years it was held to be due to an infection spreading along the nerve sheath. Weir Mitchell (1872) reported many cases of painful nerve lesions which were followed by further paralysis, sometimes in the opposite limb.

Bilateral Symmetry.—The spread of pain in a symmetrical fashion is by no means rare. In Case XVII a lesion of the left brachial plexus followed an injury to the right shoulder. In Case XXII the patient, after complaining for four weeks of pain in both shoulders, presented himself with a "winged" right scapula and profound wasting of the left spinati. Case XXIII was one of winging of the right scapula, followed a year later by severe pain in the left shoulder with wasting of the deltoid, infraspinatus and biceps. In Case XXIV neuritis of the lateral cutaneous nerves of both thighs came with a backache which followed a spell of severe military training. In Case III long-standing pain in both hands was cured when a lesion in the left forearm had been eliminated by the use of procaine.

Cases like the above have been recorded from time to time. Spillane reported bilateral lesions of the shoulder girdle in seven of his forty-seven cases; in Richardson's series the proportion was two in nine. Paget (1876) held that these instances of symmetry could be explained upon no other basis than a spread through nervous impulses. Livingston (1943) holds the same view. That such paths exist is suggested by Toennies, who, on stimulating the saphenous nerve of a cat, recorded antidromic impulses in its opposite fellow. Greenman (1913) observed that, after cutting a saphenous nerve, the number of axones on the opposite side became considerably reduced. Ray and Wolff (1945) noticed the spread of pain to the opposite side in two patients who had been subjected to unilateral chordotomy; in one case deep pressure upon the painless right leg gave rise to pain in the corresponding region of the left leg.

The Prognosis.

The recovery after a lesion in continuity usually is expeditious and complete. But the outlook in interstitial neuritis sometimes is not so good. Bennett (1939) said that about 20% of 115 patients with horse-serum neuritis were left with residual motor or sensory loss. Spillane found that the outlook often was poor in neuritis of the shoulder girdle; whereas all of Clausen's nine patients with post-operative brachial plexus lesions (1942) made complete recoveries. Of Ecker and Woltman's 150 patients with *meralgia paræsthetica* (1938), only a little more than half recovered within two years.

In only 39 of the present series of 51 cases characterized by motor or sensory signs was a reasonable follow-up possible. Sixteen of the patients had recovered fully in four months; in nine cases recovery was incomplete after periods varying from six months to a year, and in fourteen full recovery had not occurred from one to three years after the onset.

It seems likely that the frequently observed failure of patients with interstitial neuritis to recover is not consistent with the supposition that a localized lesion of a nerve has occurred. In some cases the wasting of muscles is so profound and so enduring that the observer is reminded of the sequelæ of poliomyelitis. It is interesting to recall that Gull (1868) regarded these effects as being due to permanent changes in the central nervous system.

A REFLEX THEORY OF NEURITIS.

In a recent article (1944) the writer presented evidence which suggests that, in certain cases of neuritis in which the causative lesion was not situated near the nerve trunk,

the sensory abnormality may be the result of an antidromic nervous reflex. Some evidence will now be presented which suggests a similar explanation for all the manifestations of interstitial neuritis. For this purpose the signs and symptoms may be grouped under three headings: (*a*) pain and tenderness, (*b*) alterations of cutaneous sensation, (*c*) muscular wasting, paresis and loss of deep reflexes.

Pain and Tenderness.

The reflex nature of pain and tenderness has been discussed at some length, and repetition is unnecessary.

Alterations of Cutaneous Sensation.

Abnormal Subjective Sensations.

Feelings of numbness, tingling, heat or cold may result from nerve lesions. Numbness, said Rowbotham (1939), is a very outspoken sensation, embracing coldness and swelling. In slight deviations from normal sensory function, we often have to rely upon information given by the patient. Sometimes an intelligent patient can map out an area of abnormal sensation, which to standard tests gives normal responses. A subjective sensation of numbness sometimes accompanies deep lesions; thus the pain of a sprain or earache may have a numbing quality.

Tingling, said Lewis, Pickering and Rothschild (1931), indicates interference with touch fibres. Bazett and McGlone (1932) hold that the abnormal sensations of warmth, which occur upon release of a tourniquet, are the chemical effect of accumulated products upon thermal receptors. In the writer's own case, a blow upon the ulnar nerve was followed by a vivid feeling of warmth in the hand, clearly the result of damage to fibres which convey heat sensation.

When subjective sensory disorders occur in interstitial neuritis, they are thought to indicate the presence of a nerve lesion. Tingling, however, sometimes occurs in rheumatic disease with purely muscular lesions (Ray, 1934; Judovich). In some cases of the present series tingling in the fingers was abolished simultaneously with the relief of pain by local analgesia.

Sometimes it is difficult to define where the abnormal sensations cease to be purely subjective, and where objective signs begin. Probably the difference is quantitative rather than qualitative; in other words, subjective sensations become objective when the causative process is intensified.

Objective Sensory Changes.

Lewis demonstrated that reflex hyperalgesia could involve the distribution of a cutaneous nerve through reflex antidromic stimulation. While producing artificial deep lesions, Lewis sometimes observed cutaneous hyperalgesia, which persisted for some hours, and whose distribution did not correspond with the deep component. This, he thought, was an additional effect of the deep lesion, the reflex impulses overflowing from the deep into the cutaneous pain system.

Thus the concept of reflex hyperalgesia is familiar; but the concept of the reflex production of total anæsthesia is less familiar. In neurology, however, it is recognized that there is no fundamental difference between hyperalgesia and anæsthesia; they often represent two stages of the same process (Kinnier Wilson, 1927). The observations of Adrian, Catell and Hoagland indicate that physiological impulses from touch receptors may produce, by axone reflexes, a condition of diminished activity in adjacent receptors. By stimulating a peripheral nerve, Thompson was able to produce anæsthesia in its distribution.

Earlier reference has been made to the irregular areas of anæsthesia which sometimes accompany post-traumatic dystrophy of an extremity. In some cases Miller and de Takats observed that such a patch corresponded closely with the distribution of a cutaneous nerve. This seems to indicate that the sensory effects of deep lesions are sometimes confined to the area supplied by a nerve, and sometimes more diffusely spread.

In Case XXV of the present series, the ear, and the skin within a radius of two inches surrounding it, were anæsthetic after earache lasting three days. In Case XXVI a painful sprain of the external lateral ligament of the knee generated an area of anæsthesia surrounding the limb and extending above and below the knee for four inches. In Case XXX an injury to the metacarpophalangeal joint of the thumb resulted in the appearance a few weeks later of anæsthesia over the palmar and dorsal surfaces of the thumb and forefinger, extending proximally to the radiocarpal joint both in front and behind.

Cases XXXI, XXXII and XXXIII are instances of neuritis of the lateral cutaneous nerve of the thigh (*meralgia paræsthetica*). In one the anæsthesia came on a few weeks after a sprain of the knee, and in another soon after the patient sustained a fracture of the lower end of the femur. In neither case was there any likelihood that the nerve had been injured. The reflex hypothesis seems to be applicable to them; but it receives special support from Case XXXI.

A soldier complained of painful lumps on his abdomen and thigh, with a burning ache in the thigh. On examination, four subcutaneous lipomata, about one inch in diameter, were seen on the abdominal wall. Each was tender to pressure, and surrounded by hyperalgesic skin. On the lower part of the antero-lateral aspect of the thigh was a cluster of seven lipomata, slightly larger than those on the abdomen. A large continuous area of hyperalgesia was mapped out, spreading from the iliac crest to the line of the knee joint, and occupying the whole of the anterior and lateral surfaces of the thigh.

In this case no explanation other than a reflex one seems possible. In the case of the individual lipomata, each was responsible for a small area of hyperalgesia, which has been shown by Lewis to be a reflex phenomenon. On the thigh, where the lipomata were larger, more numerous and clustered together, the areas of hyperalgesia had enlarged and coalesced, until the whole of the distribution of the lateral cutaneous nerve was occupied.

In Case XXXV, following a fracture of the lower end of the radius (which had united in a poor position), a soldier complained of weakness and numbness of the arm. Seven months later, examination revealed wasting of the muscles of the forearm, and anæsthesia of all of the skin of the arm and part of that of the chest.

When sensory changes in the skin result from deep somatic lesions, irregular patches may be affected which do not correspond with the known anatomy of nerves, or they may be confined within the field of a nerve trunk or root. When the distribution is atypical, it may involve portions of the fields of more than one dorsal root. The area involved would depend upon several factors, an important one being the integrating mechanisms in the cord which direct and coordinate the flow of impulses. Were these mechanisms to become disordered, impulses might spread in an abnormal fashion, involving ever wider fields.

Recent research has provided a partial explanation of the ease with which sensory disorders may spread from one nerve root to another (Barron and Matthews, 1935; Ranson, 1943). The proximally directed axone from the root ganglion cell gives off in the cord a great number of collaterals which make innumerable internuncial connexions on the same and the opposite side. "The pathological irritations of nerves", wrote Weir Mitchell, "may travel in either direction and influence abnormally the tissues in which lie their ultimate filaments." Charcot (1877) recorded that Samuel, by stimulating the Gasserian ganglion of a rabbit continuously for twenty-four hours, produced hyperalgesia and hyperæmia in the eye of the same side. It is impracticable, however, to reproduce experimentally the conditions occurring in disease, when the damaged tissues give off abnormal stimuli continuously for long periods.

Paralysis and Wasting of Muscles.

Wasting and weakness of muscles and loss of deep reflexes are signs of interference with the lower motor neurone. The presence of any of these signs in interstitial neuritis is accepted as further evidence of a lesion of the nerve. Wasting, however, often is seen in joint disease and with fractures, and sometimes even with contusions of muscles. In joint disease reflex activity usually is

increased rather than diminished. A denervated muscle displays excessive activity in the form of fibrillation, which persists until atrophy occurs or until nervous control is restored.

Only occasionally, however, is complete paralysis seen in interstitial neuritis. Tenderness is a common sign in fibrositis, and may be the basis of weakness or limitation of movement. The muscular stiffness is generally regarded as evidence of a somatic lesion, but the absence or diminution of deep reflexes is accepted as diagnostic of an organic lesion of the lower motor neurone. Recently, however, Gellhorn and Thompson (1944) observed that the triceps jerk disappeared with the onset of ischæmic pain in the muscle, to reappear with the restoration of the blood flow. Perhaps, therefore, diminution of deep reflexes results sometimes from conditions within the muscle; and it should not be surprising if the loss of deep reflexes is an effect of a deep somatic lesion.

Weddell, Feinstein and Pattle (1944) have reported that the wasted muscles of neuritis give varying electromyographic responses, depending upon the degree of denervation. Disordered and increased activity could be recorded from the areas of muscular tenderness in neuritis and fibrositis. Elliott (1944) observed similar activity in the areas of transient tenderness which accompanied an artificial deep lesion.

The state of the deep reflexes does not always correspond with the amount of wasting. In "sciatica", for instance, the ankle jerk may be elicited from wasted calf muscles; while in another case the reflex is absent though the muscles show no wasting. Again, loss of power is not invariably proportional to the degree of wasting. The problems presented by these inconsistencies have yet to be solved; when the nervous control of a muscle is disturbed, the details of the resulting picture cannot be predicted with certitude. In practice it is simpler to think of the muscular signs as a unit whose varying details are less important. Wasting, weakness, incoordination and disturbed reflex activity all are signs of deranged neural control, and may be present in varying degrees.

When a lesion causes widespread sensory effects, it should not be surprising if those effects overflow into the motor side. Derangement of the flow along motor nerves could bring about alterations in the deep reflexes (which are myotatic or stretch reflexes). Similarly, wasting may result from reflex nervous effects. "It is not a mere wasting from disuse", wrote Paget (1879) on arthritic muscular atrophy, "it is far more rapid than that. . . . I wish I could explain it better than by calling it reflex atrophy. It seems dependent on disordered nervous influence, and often appears proportionate to the coincident pain." Brown-Séquard (1860) was the first to suggest that the mechanism was a reflex one.

Harding (1929) showed that the wasting which accompanied experimental arthritis in rabbits did not occur if the limb had been deafferented by section of the posterior spinal roots. Previously (1925, 1926) she had shown that this reflex wasting was expensive of energy, with an oxygen consumption double that of normal muscles.

The question has been debated many times in what way disuse atrophy differs from that of denervation and from arthritic wasting. Lippmann and Selig (1928) suggested that a reflex inhibition must occur, rendering the wasting analogous to disuse atrophy. To the present writer it seems self-evident that all the different kinds of atrophy result from alterations in the flow of reflex and voluntary impulses. In denervation it is more profound because of the complete interruption of the flow. Thomsen and Luco (1944) recently made the interesting observation that the wasting which occurs with immobilization depends on the degree of relaxation of the muscles, and therefore upon the position in which the joint is immobilized. Muscles which were kept on the stretch actually gained weight while immobilized.

The atrophy of joint disease affects muscles functionally related to the joint, which have not a common supply either from peripheral nerves or from nerve roots. The spread of the impulses thus must depend upon a grouping based upon function, through internuncial mechanisms in the cord. An analogy is suggested by the cases of widespread dysæsthesia due to joint lesions, in which the manifestations bear no relation to peripheral nervous distribution.

The muscular signs in interstitial neuritis commonly follow a nerve trunk pattern, often being limited to the distribution of a single nerve or root. Often, however, portions of more than one root are involved, and a number of muscles may be picked out in a haphazard fashion. In addition to the paralysis of one or two muscles, non-paralytic wasting of a more general kind may affect most of the muscles of the region.

The Causation of Interstitial Neuritis.

Clinical Ætiological Factors.

Of Spillane's forty-six cases, twenty-six occurred in patients under treatment for wounds and for such systemic diseases as dysentery, malaria and hepatitis. Injury was responsible in two of the nine cases of Burnard and Fox, and infectious diseases in several others. Of 150 cases of *meralgia paræsthetica* reported by Ecker and Woltman, local causes were responsible in 61.

The present series is of 141 cases in which a diagnosis of interstitial neuritis could have been made; in sixty-three of these signs suggesting a nerve lesion were present. For the purpose of this ætiological study, forty-six patients with "sciatica" have been excluded, twelve of whom had motor or sensory signs. In thirty-nine of the remaining fifty-one cases the onset could be related clearly to a causative factor. In twenty-eight the cause was local, while in the remaining eleven the neuritis followed either systemic disease or damage to a distant part. Of the local causes, trauma or muscle strain was the agent in nineteen cases, *herpes zoster* in three, disease of a neighbouring joint in five and subcutaneous lipomata in one.

Local abnormality of the tissues thus plays a large part in the causation of interstitial neuritis. Of the cases due to trauma, in the majority the nerve could not, because of its site, have been involved in the primary damage. In others the signs were delayed for several days after the injury, suggesting that the trunk could not have been injured primarily. If not, it is necessary to inquire if it could have been involved secondarily in a progressive pathological condition.

In fourteen out of twenty-eight cases in which the cause was local, the manifestations had spread so that they could not be explained upon the basis of damage to a single nerve trunk. This form of spread has been known as post-traumatic spreading neuralgia (Leriche) or ascending neuritis (Walshe, 1919). Widely spreading disorders have been known to follow minor injuries, such as a too-vigorous handshake (Critchley, 1935), a slight scald of the foot (Dana, 1911) or a blow on the heel (Walshe).

Neuritis following Disease in Other Parts of the Body.

In six cases the neuritis followed an infectious disease; this was cerebro-spinal meningitis in three cases, as follows: (i) *Meralgia paræsthetica*; no recovery occurred in two years. (ii) Ulnar neuritis; no recovery occurred in six months. (iii) Involvement of the dorsal cutaneous branch of the radial nerve; the result is unknown. In three cases neuritis followed prophylactic inoculations; in one right ulnar neuritis followed amputation of the left foot, and in one brachial neuritis followed an injury to the opposite shoulder.

The spread of nervous disturbances was studied by Weir Mitchell under the name "reflex paralysis". His views were kept alive by many French neurologists, including Babinski and Froment (1918), but the conception practically died out of English medical literature under the vigorous blows of Hurst (1919), who held that all of the cases were of hysterical origin. Livingston, a modern disciple of Mitchell, lays particular emphasis on the tendency to spread displayed by many sensory disorders. The reflex disturbance", he writes, "first spreads beyond the distribution of the sensory nerve originally involved by the lesion; then it may spread to the opposite limb, to an ipsolateral limb, or even to affect the functioning of one side of the

body. . . . Once a new area is involved, the process may continue after the original lesion has lost its sustaining effect. It is difficult to escape the conviction that some dynamic process has been initiated within the spinal cord that may persist after the original stimulus has been withdrawn."

Post-Injection Neuritis.

The subject of post-injection neuritis was reviewed recently by the present writer (Kelly, 1946). When neuritis follows an injection of horse serum, its onset usually is preceded by an urticarial eruption. When a vaccine has been the cause, generalized rheumatic pains occur instead of "serum sickness". In the present series, *meralgia paræsthetica* followed serum disease due to an injection of antitetanic serum; and in two cases multiple pains followed "T.A.B." vaccination, settling later in one limb, with evidence of neuritis.

Paralyses have been reported to follow the injection of a number of different substances—milk, quinine, sulphonamides *et cetera*. The accepted theory of direct injury to a nerve trunk seems to be discounted by the following considerations: (i) Signs of paralysis usually did not appear until two days had elapsed—in one case several weeks (Elkington, 1942). (ii) In one case the paralysis was not in the same limb (Zeckel, 1939), and in the case of Sambrook (1942) the injection could not have damaged the nerve. (iii) The accepted theory assumes, in the cases of delay, a flow of material along fascial planes to reach the nerve and damage its sheath. The minute quantities injected, however, render such a supposition unlikely.

Painful Scars.

Where radiating pain is associated with a scar, it may be thought that a nerve has become involved. It is difficult, however, to see how a scar can affect a nerve in such a manner as to produce symptoms; and in any event pain is not a symptom of nerve involvement. Weir Mitchell held that some other factor was necessary to explain the syndrome. To the writer it seems more likely that the pain is related to a fibrositic lesion; it should be remembered that scar tissue is new fibrous tissue, and that fibrous tissue is the main location of the pain receptors. In a few cases impressive cures have been secured by a single injection of procaine.

Interstitial Neuritis—Primary or Secondary.

It should be emphasized again that the term "interstitial neuritis" is being used here only as a description of a syndrome which presents a fairly uniform pattern, though varying a good deal from case to case. Its pathology is not known, and it is of varied causation.

A convenient primary classification involves the separation of the cases of known from those of unknown causation. In twelve of the fifty-one cases under review, no ætiological factor could be traced; but, allowing for individual variations, the clinical picture did not show any significant difference from those which followed injury or disease. The latter group can be called secondary, and those of unknown causation primary interstitial neuritis. In speaking of "known" causation, however, no claim is made to a knowledge of the mechanism whereby the "complex" is produced; one merely knows that the mechanism was set into operation by a certain event.

When neuritis follows injury or disease, a new factor—itself the effect of the injury—comes into operation after a variable interval, and becomes independent of the exciting cause and takes charge of the situation. The clinical picture thus takes its features from the new factor rather than from the injury. In the cases of unknown causation, there usually is a history of rheumatic pain, followed after an interval by motor or sensory signs. Perineural fibrositis is generally held to be a rheumatic manifestation; the fibrositic process spreads from adjacent fibrous planes to involve the perineurium. The fully developed picture in primary or rheumatic neuritis does not differ fundamentally from that of the secondary cases, because the same unknown factor has come into operation, though without any discernible exciting cause.

In both primary and secondary cases, pain and tenderness have been abolished by anæsthetizing deep lesions, and the evidence suggests that the diffuse tenderness is a reflex effect. Burnard and Fox obtained a similar result in one of their cases. In previous essays the present writer has demonstrated that the features of fibrositic lesion do not present any significant variation, whether its cause is known or unknown. The pain and tenderness being reflex effects from a fibrositic lesion, it is suggested that the motor and sensory signs (which, it has been shown, may be reflex effects) also have been set in motion by the same lesion. The unknown factor which is responsible for the manifestations of neuritis closely resembles a fibrositic lesion in its behaviour.

Functional Nervous Signs.

The Functional Organization of the Cord.

The tendency of the orthodox view is to dismiss as a psychic phenomenon every sensory abnormality which cannot be explained as the effect of an organic lesion interfering with nerve trunks or nerve tracts. Very little, however, is known about the functional organization of the cord as it affects sensation. The motor organization, we know, has as its basis movements rather than individual muscles or individual segments of the cord (Sherrington, 1906). Thus a simple reflex movement requires a complicated form of coordination which sometimes spreads over a large number of segments of the cord, bringing about the stimulation of some groups of muscles and the inhibition of others. It seems possible that sensory impulses may be subject to an analogous form of coordinated spread: indeed the large numbers of collaterals which proceed in all directions from sensory radicles would seem to imply some sort of interlinkage between sensory neurones. And the fact that some such sensory reflex organization exists is now well established. The work of Barron and Matthews (1935) has been confirmed in its important details by Toennies (1938), Barron (1940) and Hursh (1940). In addition to the normal sensory impulses which ascend by the sensory tracts to the cerebrum, delayed discharges of a reflex nature can be recorded, which spread both upwards in the cord and outwards through the posterior roots. "The sum of the evidence now available", writes Hursh, "indicates that an afferent volley, whether artificially excited or made up of physiologically selected sensory impulses, after a central reflex time of 2·1 to 2·6 millisecs., evokes a reflex in dorsal root fibres. At the same time that the reflex discharge passes outward to the periphery, the discharge is also carried along the central branches of the dorsal root fibres toward the nuclei of Goll and Burdach."

"The Nervous System", said Lorente de Nó (1938), "is composed of an exceedingly large number of interlacing pathways which offer numerous opportunities for the conduction of impulses into divergent paths; but during activity it becomes fractionated into a group of active and a group of inactive neurons." In other words, stimulation and inhibition are continually proceeding side by side in the spinal cord, both on the motor and on the sensory side. It seems not unreasonable, therefore, to suggest that a succession of abnormal sensory impulses, such as may proceed from diseased or damaged tissue, could set up in the cord a disorder of function, a functional disturbance in the truest sense of the word, which would manifest itself by abnormal sensory or motor phenomena.

Hysterical or Actual?

When sensory changes do not correspond with known anatomy of nerves, it is usual for hysteria to be diagnosed. Ill-understood bodily reactions to trauma have come to be associated (in the medical mind) with disordered emotional states. In Charcot's celebrated cases of hysteria the patients displayed unpredictable somatic effects, and abnormal reactions could be produced under hypnosis. Charcot himself maintained a clear line of demarcation between the psychopathic and the "physiopathic", but this distinction was not maintained by many of his disciples. Babinski produced strong reasons for recognizing an organic basis for the signs in such cases as have been

described in this paper, but in English neurology the views of Hurst came to prevail. It is normal to regard signs which cannot be explained as unreal, a reproach to the patient; more especially if a history can be obtained of a grievance, an unpaid compensation claim or an abnormal emotional state.

The diagnosis of hysteria is of that "dead-end" kind which blocks inquiry; when once it has been made, further investigation is forbidden as likely to "fix" the patient's symptoms by suggestion, and the only form of inquiry left is of a psychiatric nature. Codman (1934), who devoted many years to the study of painful shoulders, wrote: "I have the feeling that some neurologists, who have seen many mental cases, are too ready to pin the label of hysteria on symptoms which may possibly be due to physical conditions of which they are ignorant."

When one is confronted with signs which cannot be explained upon the basis of present knowledge, the temptation to explain them away as functional is very great. In so far, however, as they are due to reflex disturbances of function, no more accurate term than "functional" can be applied to them. In popular medical usage, however, the expression has not this significance. "In the present conflict", wrote W. K. Livingston, "surgeons all over the world will be confronted by cases that exhibit sensory changes and the loss of motor function so bizarre in their distribution, and apparently so unrelated to the apparent causes as to throw doubt on there being any possible organic basis for them. . . . They will observe functional and trophic disturbances of wide distribution that may not conform to any pattern of nerve distribution which their textbooks had taught them to expect. Because of these things there will be a great temptation to deny to such cases an organic basis, and to ascribe the symptoms to psychic causes for which the patient may in some way be responsible."

SUMMARY.

1. The term neuralgia covers a variety of painful conditions, in which the pain and other signs are the effects of nervous reflexes, set in motion from a painful focus and acting upon somatic tissues by means of impulses travelling antidromically in sensory nerves.

2. In interstitial neuritis, on the other hand, the main lesion is thought to involve a large nerve trunk, and to produce pain and motor and sensory disorders by pressing on axis cylinders. Of this there is no pathological proof. All authorities agree that pain and deep tenderness are the main symptoms of interstitial neuritis, and that motor and sensory signs occur in only a minority of cases.

3. The nature of somatic pain and deep tenderness is discussed in the light of recent research. A review of our knowledge of the behaviour of nerve fibres, and of the clinical effects of lesions of nerve trunks, indicates that pain is not a characteristic effect of interference with axis cylinders. In addition, the picture of interstitial neuritis differs from that of a traumatic lesion in continuity on several other important points.

4. Recent research upon fibrositis (which is not a well-defined pathological entity) suggests that the pain and tenderness are the result of nervous reflexes initiated by a focal lesion. Observations made by the present writer agree with this. While investigating many cases of fibrositis with local analgesia, the writer had under observation a large number to which the clinical diagnosis of interstitial neuritis could be applied. In the majority it was possible, by injecting procaine into fibrositic lesions, to abolish the pain and the deep tenderness either permanently or for the duration of the analgesia.

5. In none of these cases was paralysis of nerves caused by the injection. The focal points of deep tenderness therefore could not have been closely related to any important nerve trunks, and it is suggested that "interstitial neuritis", when it does not present signs of a nerve lesion, cannot be differentiated from fibrositis.

6. Cutaneous anæsthesia and muscular paresis, when they occur, can also be explained as the effect of disordered nervous reflexes originating in a fibrositic lesion. Recent research in neurophysiology suggests that such a mechanism exists.

7. An ætiological study indicates that interstitial neuritis, in the majority of cases, is brought on by tissue damage or disease. A new factor appears to take control which becomes independent of the exciting cause. Whether the cause is known or unknown, the clinical picture shows no significant variation.

8. These painful disorders of the peripheral nervous system have an organic basis, and it is an error to think of them as hysterical.

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THE INFLUENCE OF ANÆSTHETICS ON THE CONTENT OF DEAMINATING ENZYME IN HUMAN PLASMA.

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It has been shown by Drury, Lutwak-Mann and Solandt[1] (1938) that human plasma contains an agent capable of inactivating adenyl compounds. These workers have also shown that in the cat following ether anæsthesia and decapitation there is a significant increase in the deaminating enzyme activity of the plasma over a period of some hours. It was concluded that a number of factors other than the anæsthetic were either wholly or in part responsible for the increased activity. Since it has been shown

that chloroform (Trethewie,[4] 1941) causes the liberation of deaminating enzyme from the perfused liver and of adenyl compounds from the perfused and intact liver of the dog, it was thought worth while to investigate the influence of anæsthetics on the content of this enzyme in the plasma of human subjects submitted to operation.

Method.

The method used was that described in an earlier publication (Trethewie,[6] 1942). Lacarnol was added to heparinized plasma to make a solution of one part lacarnol to 50 parts system; a 4·6% concentration of carbon dioxide in oxygen was played over the surface of the plasma continuously after it had bubbled through water. Samples of plasma were diluted and boiled prior to being tested. The notation used to indicate the enzyme content of the plasma is the percentage of cardio-inhibitory activity remaining after forty minutes' incubation at 39° C. (±0·5° C.) and a pH of 7·5 to 7·9. Repeat tests of any one sample of plasma were carried out with a pH within 0·08 of the earlier test. As has been recorded earlier (Trethewie),[3] such a variation in pH does not significantly affect the deaminating activity of the plasma. Since the pH lay always in this range when it was tested, some of the later tests were made without its being determined. Cardio-inhibitory activity was assayed by the method of Drury, Lutwak-Mann and Solandt.[1]

Experimental Investigation.

The Content of Inactivating Enzyme in Human Plasma.

Drury, Lutwak-Mann and Solandt[1] have shown that when adenosine is mixed with human plasma there is a progressive fall in cardio-depressant activity of the system following incubation. In these experiments we have used lacarnol (Bayer), a tissue extract containing adenyl compounds instead of adenosine. Similar results were obtained. Twenty estimates were made prior to operation on patients who were afebrile and whose conditions were one of the following: hernia, long-standing fracture (six weeks or more), chronic joint condition (patient afebrile), long-standing (six weeks or more) recovery from appendicitis, gland biopsy, prostatomegaly, and operable malignant disease of the bowel. After forty minutes' incubation of the plasma-containing lacarnol (1 in 50), the cardio-depressant activity of the mixture was reduced to 30% to 75%. The average figure was 53%. If it is desired to select the presumably healthy—that is, the first two groups above (14 patients)—the average figure is 50%. All cases, however, are included, since we are investigating the influence of anæsthetics on those patients who are submitted to surgical operation and anæsthesia. The histogram of distribution for all these cases is shown in Figure I. The portion enclosed in the dotted line represents two patients in the series who had plasma with deaminating activity equivalent to reduction of the lacarnol content to 30% in forty minutes. One had been anæsthetized two days previously, and the other was suffering from a "cold".

The Variation in Plasma Content of Enzyme over Forty-Eight Hours.

There is little variation in the plasma content of deaminating enzyme in patients kept in hospital who feel well and are not subjected to operation or anæsthesia. For example, in a case in which two samples were collected at an interval of two hours, the activities of the plasma after incubation were 62% and 60%. In another case, in which two samples were collected at an interval of four hours, the figures were 48% and 45%. In yet another case, in which the interval between collections of samples was five and a half hours, the result of the second test was 3% higher than that of the first, and in one case, in which the interval was eighteen and a half hours, there was no change. In two cases in which two tests were made at an interval of forty-eight hours, the activities of the plasma after forty minutes' incubation were 60% and 60% in the one instance and 48% and 45% in the other. These findings are shown in Table I.

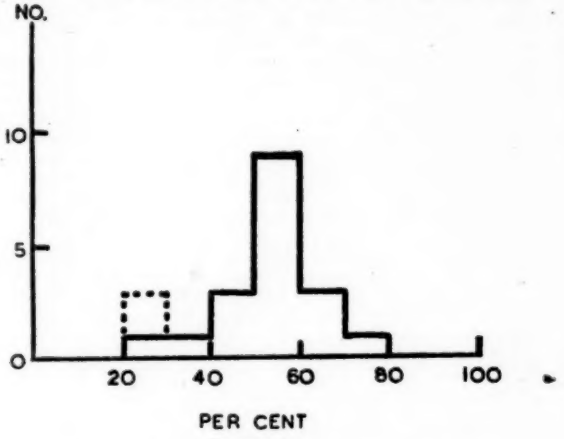

FIGURE I.

Graph showing the distribution of plasma deaminating enzyme activity in subjects prior to operation. Ordinate: number of cases in 10% groups. Abscissa: deaminating enzyme activity as percentage of lacarnol (2%) remaining active after incubation for forty minutes at 39° C.

The Change in the Content of Deaminating Enzyme following Operative Interference and Ether Anæsthesia.

After operations under ether anæsthesia there is a significant increase in the activity of the plasma, as measured by its capacity to inactivate lacarnol. These findings are tabulated in Table II. In one instance (Experiment XVIII) plasma taken twenty-five minutes prior to operation had reduced the activity of the lacarnol system to 88% in twenty minutes and to 75% in forty minutes. Plasma taken ninety minutes after the patient's anæsthesia was commenced reduced the activity of the lacarnol to 80% in twenty minutes and to 35% in forty minutes. If we denote the increased activity of the plasma after anæsthesia (which changed from 75% to 35% after forty minutes' incubation) as a percentage—namely, $\frac{75-35}{75} \times 100$—we have a figure for the increased activity of the plasma,

TABLE I.

Controls: Activity of First Sample of Plasma (i), and of Plasma at Repeat Test (ii), after an Interval without Anæsthesia.

Experiment Number.	Condition Affecting Patient.	Percentage Activity of Plasma after Incubation.		Percentage Decrease in Activity of Plasma. [(i)-(ii).]	Percentage Change. $\frac{(i)-(ii)}{(i)} \times 100$.	Time between (i) and (ii). (Hours.)
		(i)	(ii)			
I	Carcinoma (rodent ulcer)	62	60	2	3	2
II	Benign hypertrophy of the prostate	48	45	3	6	4
III	Fracture of the femur	51	54	−3	−6	5·5
IV	Fracture of the pelvis	30	30	0	0	18·5
V	Fracture of the ankle	48	45	3	6	48
VI	Chronic arthritis of the right foot..	60	60	0	0	48

which in this instance is 53%. The increase in activity of the plasma continues for some hours after operation. The continuous line in Figure II shows the variation of the activity of the plasma calculated in this way; the ordinates indicate the percentage change in activity and the abscissæ the time from the commencement of the anæsthesia (in hours or days) when the second sample of plasma was taken. It is seen that the maximum increase appears about two hours after the commencement of the anæsthesia, and after twenty hours there is a secondary rise. In one case a fall occurred when the patient had a "cold" when the first sample of blood was taken prior to operation, though this difference was insignificant. This sample was obtained three-quarters of an hour after the commencement of anæsthesia. In two instances in which blood was obtained eighteen and twenty hours after the commencement of the anæsthesia, the blood after anæsthesia also contained slightly less enzyme activity and the difference was greater than that observed in controls. In one of these difficulty was experienced in inducing anæsthesia with ether, and on the day after operation, when the second blood sample was taken, the patient was extremely well. Anæsthesia was light. In the other some hæmolysis was detected in the first sample, and this probably explains the lower figure obtained for this specimen (see section on the effect of hæmolysis on deaminating enzyme activity). For this reason this experiment (Experiment XVI) is excluded from Figure II. The interrupted line (Figure II) shows a negligible change in the enzyme content in control cases in which no operation was performed or anæsthesia administered. In Experiment VIII (Table II) some hæmolysis was detected in the second specimen; this probably contributed to the increase in enzyme activity of this sample, and therefore it is excluded from Figure II. Experiment XII is included in Figure II, since any effect of hæmolysis (in the first sample) is to decrease the difference in activity of the plasma, which is still considerable. Except for this experiment there was no hæmolysis in any other of the samples included in the figure.

This increase in enzyme activity is not a direct effect of ether on the plasma, since the addition of ether to plasma in the concentration found during anæsthesia does not affect the rate of deamination. For example, in one experiment the activity of a sample of plasma was reduced to 55% after forty minutes' incubation, and in the case of another sample of the same plasma to which ether was added (2·3 grammes per litre of plasma), the activity was reduced to 53% after incubation for the same time.

Since it is difficult to compare the changed activity of enzyme in the plasma of different patients, and though when this was attempted, the importance of the concentration of the lacarnol left at the end of forty minutes (A) has been taken into account—that is, the normal activity

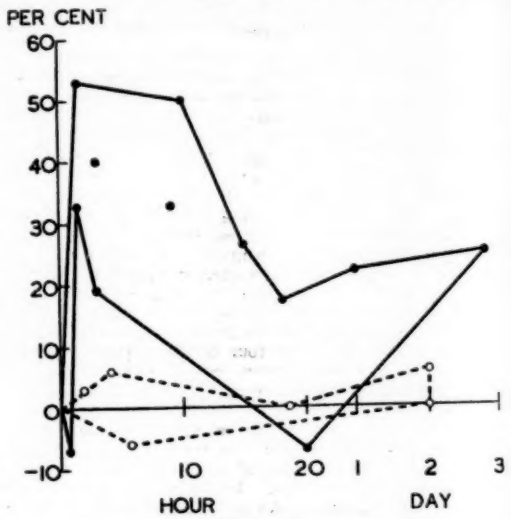

FIGURE II.

Continuous line: change in enzyme activity ($\frac{A-B}{A} \times 100$) of plasma after ether anæsthesia. Interrupted line: change of enzyme activity of plasma without anæsthesia. Ordinate: percentage change of activity. Abscissa: hours and days after beginning of anæsthesia when second sample was taken. Further details in text.

of the enzyme for the particular patient—by assessment of the change in activity as a percentage ($\frac{A-B}{A} \times 100$, where B is the activity after incubation following ether anæsthesia), a figure has been drawn showing the linear change in activity of the enzyme as measured by difference of lacarnol percentage remaining active after incubation ($A - B$; in 10% groups) and the number in each group.

TABLE II.

Activity of Plasma (A) before and (B) after Ether Anæsthesia.

Experiment Number.	Condition Affecting Patient.	Operation.	Percentage Activity of Plasma after Incubation.		Percentage Decrease in Activity of Plasma. ($A-B$.)	Percentage Change. $\frac{(A-B)}{A}$	Time from Beginning of Operation when Sample B was Collected. (Hours.)	Comment.
			A.	B.				
VII	Hernia (indirect inguinal).	Bassini.	30	32	−2	−7	0·75	"Cold" before operation.
VIII	Hernia (inguinal).	Hernial repair.	53	15 (hæmolysis)	38	72	2·5	
IX	Hernia (inguinal).	Bassini.	45	30	15	33	9	
X	Hernia (incisional).	Repair of hernia.	70	75	−5	−7	20	Very difficult to anæsthetize. Patient very well. Takes alcohol.
XI	Lump in groin.	Biopsy.	60	47	13	22	24	
XII	Carcinoma (rectum).	Colostomy.	70 (hæmolysis)	47	23	33	1·5	
XIII	Carcinoma (stomach).	Pólya.	63	38	25	40	3	
XIV	Carcinoma (rectum).	Colostomy.	37	30 (vomiting)	7	19	3	
XV	Carcinoma (rectum).	Colostomy.	55	50	5	9	7	
XVI	Carcinoma (colon).	Abdominal tumour removed.	50 (hæmolysis)	60	−10	−20	18	
XVII	Appendicitis (subacute).	Appendicectomy.	60	45	15	25	67	
XVIII	Fracture (radius and ulna).	Open reduction.	75	35	40	53	1·5	Fracture one month before A.
XIX	Fracture (radius and ulna).	Manipulation.	58	43	15	26	15	
XX	Fracture (radius).	Open reduction.	30	25	5	17	18	Anæsthetic few days before A.
XXI	Cholelithiasis.	Cholecystectomy.	20	10	10	50	10	

This is shown in Figure III. As before, Experiments VIII and XVI are excluded because of hæmolysis. It is noted that in all of the control cases (interrupted line) the value is ± 5%. There is a shift to the right in the cases of ether anæsthesia. Variables not indicated in the figure are the amount of ether absorbed and the degree of trauma at operation. The time (in hours) from the commencement of operation when sample *B* was taken is indicated in Figure III.

The Change in the Content of Deaminating Enzyme following Operative Interference and Spinal Anæsthesia.

In contradistinction to the effect of a volatile anæsthetic (ether), when patients are operated on under spinal anæsthesia there is no significant change in the enzyme activity of the plasma, provided no difficulty is experienced at operation. For example, in one case in which a patient was subjected to operation (suprapubic prostatectomy), the blood before operation contained an enzyme activity equivalent to reduction of the adenyl compound content of the plasma to 53% after forty minutes' incubation, and two hours after operation the corresponding figure was 55%. These findings are shown in Table III. It is noted that there was negligible variation in four of these cases. In one, a case of intestinal obstruction, considerable trauma was produced during operation owing to the fixity of the bowel, and there was an increase in the enzyme content of the blood two and a quarter hours after the beginning of operation. The condition of this patient was poor and he died. The vein was occluded for some time before the second sample was collected. In the other cases, in which simple cystoscopy was performed, there was no significant change in the enzyme content of the plasma two and a half hours after anæsthesia.

For comparison, Table IV includes patients operated on under ether and spinal anæsthesia, from whom samples of blood were tested one and a half to three hours after the operation was commenced. In patients operated on under ether anæsthsia, at one and a half to three hours, whether the condition is hernia, bowel abnormality or fracture, there is always a considerable increase in the enzyme activity of the plasma (19% to 53%). Of four patients subjected to operation under spinal anæsthesia at this time, in three there was no significant change (−9% to +9%); in the other case, in which there was an increase in the enzyme activity (55%), the patient was very ill and died after a difficult operation.

The Effect of Hæmolysis on Deaminating Enzyme Activity.

In two cases operated on under spinal anæsthesia there was an increase in the enzyme content of the plasma after operation, but in each the blood obtained after operation was slightly hæmolysed and the blood prior to operation was not. These findings are shown in Table V. In Experiment XXVIII the plasma contained 230 milligrammes *per centum* of hæmoglobin. Since similar operations under spinal anæsthesia were not associated with an increase in enzyme activity, and since in these latter no hæmolysis was noted, it was thought worth while to determine whether hæmolysis itself was associated with an increase in enzyme activity. Such was the case. Plasma was obtained from a patient, and to one part (2·05 millilitres) 1·1 millilitres of distilled water and 0·33 millilitre of lacarnol (diluted 1/5) were added, and to the other part (2·05 millilitres) 1·0 millilitre of distilled water containing 0·1 millilitre of packed cells was added and as before 0·33 millilitre of lacarnol (diluted 1/5). The hæmoglobin content of this mixture was 0·8 gramme per 100 millilitres. The content of the control was estimated as 1/50 lacarnol (100%) when the mixture was boiled immediately. The content of the part to which hæmoglobin was added was estimated as 113% when it was boiled immediately. This rise was probably due to the fact that red cells contain adenyl compounds. After incubation for forty minutes the control contained 68% activity lacarnol and that to which hæmoglobin was added only 50%. The plasma had a lower activity than usual because it was diluted 40%. Thus there was an increase in enzyme activity with the added hæmoglobin.

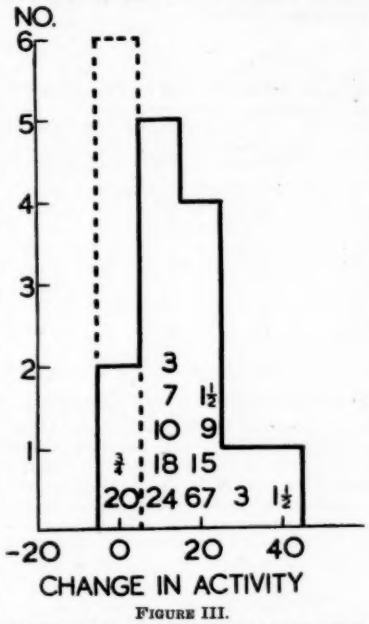

FIGURE III.

Increase in enzyme activity (*A* − *B*) after ether anæsthesia. Ordinate: number of cases in groups −5 to +4, 5 to 14, 15 to 24, 25 to 34. Abscissa: difference in enzyme activity of plasma after and before anæsthesia. Continuous line: ether anæsthesia. Interrupted line: controls.

TABLE III.

Activity of Plasma, (A) before and (B) after Spinal Anæsthesia.

Experiment Number.	Condition Affecting Patient.	Anæsthetic Agent.	Operation.	Percentage Activity of Plasma after Incubation.		Percentage Decrease Activity of Plasma. (*A* − *B*).	Time from Beginning of Operation when Sample *B* was Collected. (Hours.)	Comment.
				A.	*B*.			
XXII	Benign hypertrophy of the prostate.	Percaine.	Prostatectomy.	53	55	−2	2	
XXIII	Benign hypertrophy of the prostate and diverticulum (bladder).	Light percaine.	Excision of diverticulum.	55	50	5	2	
XXIV	Ureteric calculus.	Heavy percaine.	Cystoscopy.	42	46	−4	2·5	
XXV	Benign hypertrophy of the prostate.	Heavy percaine.	Prostatectomy.	40	43	−3	19	
XXVI	Benign hypertrophy of the prostate.	Light percaine.	Prostatectomy.	52	52	0	19·5	
XXVII	Carcinoma of the colon (obstruction).	Heavy percaine.	Colostomy.	33	15	18	2·25	Much mobilization required. Patient's condition poor. Died. Vein occluded some time before sample *B* was collected.

Included in Table V is a case of orchidectomy in which operation was performed under local anæsthesia and in which hæmolysis was present in the first sample of plasma; this was the sample which showed increased enzyme activity (in fact, it was the most active sample of plasma ever obtained prior to operation). In a patient (Experiment XXXI) who had received only an injection of local anæsthetic, there was no change in the enzyme content before and after the procedure (Table V).

TABLE IV.

Change in Enzyme Activity $\left(\frac{A-B}{A}\times 100\right)$ *of Plasma after Ether and Spinal Anæsthesia.*

Operation.	Anæsthesia.	Percentage Change.	Time. (Hours.)	Comment.
Open reduction of fracture.	Ether.	53	1·5	
Colostomy ..	Ether.	33	1·5	
Colostomy ..	Ether.	19	3	
Gastric resection	Ether.	40	3	
Prostatectomy ..	Spinal.	−4	2	
Bladder diverticulectomy.	Spinal.	9	2	
Colostomy ..	Spinal.	55	2·25	Died.
Cystoscopy ..	Spinal.	−9	2·5	

The Effect of Local Urethral Anæsthesia and Cystoscopy.

In a male patient on whom tests were made of the blood before and after cystoscopy under local urethral anæsthesia (Ryall's solution) there was no detectable change in the content of deaminating enzyme. These findings are shown in Table VI. In one instance the plasma three hours after cystoscopy was 3% higher in content of adenyl compound after incubation than before cystoscopy, in one there was no change, and in the other there was a fall of 5%.

Discussion.

It is evident from this work that under the influence of ether anæsthesia surgical procedures on man are followed by the liberation of deaminating enzyme into the blood. When a severe surgical procedure (decapitation) is undertaken on cats, there is an increase of deaminating enzyme in the blood (Drury, Lutwak-Mann and Solandt[1]), and these authors adduce that the operation or other factors are more important than the anæsthetic. In the work described here, when operations are performed under spinal anæsthesia there is usually no increase in the enzyme content of the blood. In the one instance in which such an increase occurred with spinal anæsthesia, the operation was difficult, surgical trauma was considerable, and in fact the patient died.

When we compare the type of operation performed when ether anæsthesia was used, whether the condition was simply a manipulation of a fracture, a simple herniotomy, or a bowel operation, there was always a considerable increase in enzyme activity two to four hours after the beginning of operation. On the contrary, the operation of suprapubic prostatectomy performed under spinal anæsthesia was not associated with any increase of enzyme. It must be stated that in these cases no difficulty such as hæmorrhage was encountered. Neither was such a difficulty encountered in the cases in which ether anæsthesia was used.

Therefore, on the evidence we can conclude that ether itself is responsible in man for causing an increase in the enzyme content of the blood, and since ether has not this effect *in vitro* it is possible that the effect is indirect. It has been shown in animals that chloroform, a volatile anæsthetic, causes the liberation of deaminating enzyme from the isolated perfused liver of the dog (Trethewie[4]). Ether in a concentration of 10% causes the liberation of slow-reacting, smooth-muscle stimulating substance (S.R.S.) from the isolated perfused lung of the dog (Kellaway and Trethewie,[2] 1939), and this concentration is used during induction, so that some injury at least is produced in the lung by concentrations normally used. Ether is avoided in man in cases of lung disease, and while we have no evidence of toxic effects of S.R.S., the lung contains deaminating enzyme (it is liberated in pneumonia—Trethewie[3][4]) and it may be that the enzyme is liberated from the lung under ether anæsthesia. Spinal anæsthesia might, then, not be expected to cause a liberation of such an enzyme, because it does not come into intimate contact with lung.

In pneumonia it was shown (Trethewie[3]) that deaminating enzyme was liberated into the blood when the patient was ill. With recovery the enzyme content of the blood returned to the normal level. With a recrudescence of

TABLE V.

Activity of Plasma in Relation to Hæmolysis.

Experiment Number.	Condition.	Anæsthesia.	Procedure.	Percentage Activity of Plasma after Incubation.		Percentage Decrease in Activity of Plasma. [(i)-(ii).]	Time from Beginning of Operation when Sample *B* was Collected. (Hours.)
				(i)	(ii)		
XXVIII	Benign hypertrophy of the prostate.	Spinal (light percaine).	Prostatectomy.	40	25 (hæmolysis)	15	1·5
XXIX	Benign hypertrophy of the prostate.	Spinal (light percaine).	Prostatectomy.	58	33 (hæmolysis)	25	4
XXX	Cystitis.	Nil.	Hæmolysed cells added to (ii).	68	50	18	—
XXXI	Spondylolisthesis.	Local, with "Novocain".	Injection only.	40	40	0	2·5
XXXII	Tuberculous epididymitis.	Local, with ethocaine.	Orchidectomy.	15 (hæmolysis).	25	−10	1·5

TABLE VI.

Activity of Plasma, (A) before and (B) after Local Urethral Anæsthesia.

Experiment Number.	Condition Affecting Patient.	Anæsthetic Agent.	Operation.	Percentage Activity of Plasma after Incubation.		Percentage Decrease in Activity of Plasma. (*A* − *B*.)	Time (Hours) after Operation when *B* Tested.
				A.	*B*		
XXIII	Hydronephrosis.	Ryall's solution.	Cystoscopy.	53	56	−3	3
XXXIV	Cystitis.	Ryall's solution.	Cystoscopy.	55	55	0	3·5
XXXV	Benign hypertrophy of the prostate.	Ryall's solution.	Cystoscopy.	40	35	5	4

pneumonia in one case, again the enzyme content rose to higher figures. It was suggested that the presence of the increased amount of enzyme in the blood might be related to the feeling of malaise of the patient. Now it is evident that patients subjected to operation under spinal anæsthesia are far more comfortable soon after simple operations than are patients subjected to operation under ether anæsthesia. I have questioned patients who have undergone operation for the same condition under these forms of anæsthesia and this observation has been confirmed subjectively. Usually this condition of malaise following ether anæsthesia is regarded as "the effects of the anæsthetic wearing off", and it is not for some hours that the patient begins to "feel himself" again. In contradistinction, patients who undergo operation under spinal or local anæsthesia do not pass through this phase. Now it is just during this period that there is an increase of deaminating enzyme in the blood. Potassium is liberated after ether anæsthesia (Stehle, Bourne and Barbour,[3] 1922), passing into the urine. However, when the patient is ill with pneumonia, at the onset of the disease the potassium content of the blood was low (Trethewie and Cowan,[7] 1946). Later there is a rise in the potassium level. It seems unlikely, therefore, that a rise in potassium level is an essential factor in the development of the feeling of malaise. The deaminating enzyme content is high at the onset and falls to normal with recovery. Histamine is also liberated in pneumonia, but only a fraction of that liberated passes immediately into the blood (Trethewie,[6]). It seems unlikely, therefore, that histamine accounts for the feeling of malaise in pneumonia. Further, histamine is not liberated by ether in the dog till high concentrations are reached (19% to 44%); these are not attained in man (Kellaway and Trethewie, 1939).

It is possible that many substances are responsible for the feeling of "sickness" in different conditions, in addition to nervous effects and direct effects of injurious agents; but the foregoing discussion is put forward tentatively to stimulate criticism.

Summary.

After surgical procedures under ether anæsthesia there was an increase in the deaminating enzyme content of the plasma of human subjects.

After surgical procedures under spinal anæsthesia there was no increase in deaminating enzyme content of plasma except in one instance, in which the operation was performed with difficulty and the condition of the patient was low.

Simple tests under local urethral anæsthesia showed it to be without effect on the content of deaminating enzyme of the plasma.

Hæmolysis of blood increased the deaminating enzyme activity.

The significance of these findings is discussed.

Acknowledgements.

Thanks for cooperation are due to the honorary medical staff of the Royal Adelaide Hospital and of the Royal Melbourne Hospital.

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A REPORT ON THE PSYCHOPATHIC STATES OF THE AUSTRALIAN IMPERIAL FORCE IN THE MALAYAN CAMPAIGN.

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This report was written during life as a prisoner of war at Changi in 1942, from notes preserved during the chaos of capitulation, siege and transportation from Singapore city to Changi. Difficulty in their further preservation was experienced in the searches at embarkation and disembarkation and in the intimate life in Kobe camp in Japan, where the Nipponese treated us as private soldiers in the Imperial Japanese Army and regarded every item of our scanty belongings with extreme suspicion. During a period of just over two years the notes survived numerous searches, two severe earth tremors, and finally the total extinction of Kobe camp, in a few minutes, by the incendiary bombs of our ultimate rescuers.

Part I: Neuroses.

Production of Neuroses.

> Cowardice in one form or other is visible in every human being; it is at the bottom of the whole caste system; the foundation of every organised society.
> —H. L. Mencken.

Brutally direct, Mencken exposes the principle of all neurosis production without preamble. Fear, the basis of the self-preservation instinct, is the reason for herd formation and is the dominant factor in the evolution and maintenance of the codes, the conventions, the laws and the beliefs of sociology. Fear may be physical, with thoughts of death, mutilation or organic abnormality, or it may be social because of inability to cope with a situation associated with embarrassment and indecision or with fear of ridicule from fellow members of the herd; or the fear may be fear of loss of power, of wealth or of prestige. Whatever its type or causation, fear may be concealed and hidden from the world, or it may be latent; but it is manifest in the neuroses. Exhibition of these is characterized by alterations in the appearance, the conduct and the conversation of the subject, which produce objective changes in the soma with alterations in the respiratory, cardio-vascular, digestive and other systems, and subjective effects upon the psyche with statements by the patient as to his feelings and emotions.

The sudden appearance or the noisy approach of a large object, a cry of terror or of pain by a member of the herd, or a feeling of insecurity or of falling, will produce in anyone a fear reaction or a tendency to react. As primary causes of fear these factors are evolutionary, innate and a common inheritance. All other fear-producing factors are secondary; they are acquired from the environment of the individual, subsequent to his birth; they are social influences producing fear reactions or tendencies to react, variable in the individual and in individuals; they are not common to all members of the herd.

Primary fear reactions, then, are instinctive behaviour reactions and, as such, they can be replaced or modified. Replacement or modification is necessary in everyday life to control and to inhibit instinctive tendencies in accordance with the moral and the social customs prevailing. Behaviour, to be instinctive, is necessarily devoid of thought and intelligence, of training and of example; yet such behaviour is always purposive, even though the purpose may not be patent to the performer. When fear is the impelling agent and when replacement or modification of instinctive behaviour is necessary in civilian everyday life, how much more so, then, is it necessary that replacement or modification be inculcated into the behaviour reaction pattern of the daily life and habits of a soldier? In his case training and example assume paramount proportions in the production of substitute reactions to danger stimuli. Such substitute reactions lie in the orderly move-

ments devised and planned for his self-preservation and well-being. They are driven into his character and into his conduct by constant training in discipline and in obedience, and by example.

In the breaking down of the mental structure of the soldier, with exhibition of instinctive behaviour and with neurosis production, either something must be lacking or the stress and strain operative in his environment must have been too severe or of too long duration. As it has been so tersely put by Mencken in the quotation at the beginning of this paper, it may be gathered and appreciated that occasionally environmental stress of long duration and severity will undermine the strongest character and personality. Observation of men suffering from neurosis in Malaya reveals the following facts: (i) that in the vast majority of cases—in fact, in almost all—the subject lacks ability and temperament fully to absorb the tenets of military training and example; (ii) that the stress and strain of conditions of life on active service overseas are precipitating factors only; (iii) that the primary cause lies in the constitution of the subject.

Consideration of subjects of neurosis encountered in pre-war (Malayan) conditions, during the war, and as prisoners of war, brings to light a mixed ætiology, with constitutional and precipitant factors, emerging from heredity and from the civilian social and military environments, providing food for medical and military thought.

Incidence of Neuroses.

Before the War in Malaya.

Patients numbering 165 passed through the Second/Second Australian Convalescent Depot. A number were deemed unfit and were sent home to Australia. Hysterical topical fixations and preoccupations attributed mainly to the stomach and to the head were common. Some subjects showed an admixture of hysteria and of malingering. Aphonia and limb paralyses were encountered as fixations and as conversions. Some complete martial misfits were eradicated from the division. "Tropical" or "Malayan" memory was universal, degrees of amnesia for both recent and remote events being present.

Neuroses of War.

Patients examined at the depot numbered 89, and from these 51 histories were obtained; the remainder came during the last three days of the war, when 1,100 convalescents were somehow quartered in the Cathay Theatre. Other cases occurred and the patients did not come to us; some remained in hospitals, others were held in units and treated by unit medical officers. At all times, owing to conditions of war, examination of patients, the taking and keeping of notes, and treatment were difficult. The average age of the 51 patients (and probably of all) was thirty-four years. In symptoms and ætiology there were many common features, with occasional individual differences. Treatment was by sedation (when possible), explanation, sympathy and reassurance. The results obtained were on the whole satisfactory, for even under the conditions prevailing upon Singapore island a number of patients were returned fit to units and to action. The stress and strain of active warfare in precipitating the neuroses varied in degree of severity from absent to severe and prolonged. Most frequently the precipitating factor was near bomb-burst, with no discoverable somatic lesion; some men broke down just before or at the time of going into action, many after several days of moderately severe experience, while a few, after a long and harrowing time, broke down when they had been removed from danger and to a place of quiet and rest. It was commonly stated that the conditions at the depot, whence they had been sent for treatment, were worse than those that had produced the neurosis.

Neuroses of Prisoner-of-War Life.

Under prisoner-of-war conditions only 11 cases of neurosis occurred in the first fifteen months. The average age of patients was thirty-three years. All the neuroses were depressive in character. New symptoms and ætiological factors were observed. Two subjects were suffering from pure hysteria, and these were the only ones who did not respond to treatment.

Ætiological Factors of the Neuroses.

Consideration must be given to the changes in environmental features experienced by the men; these varied from those of pre-war military life in Malaya to those encountered in a rapid mobile offensive war of sudden onset, again to those of prisoner life with starvation and new and alarming diseases and deaths, again to a twenty-three day sea voyage to Japan not devoid of incident or peril, and finally to those of prisoner life in Japan with the attendant dangers of Allied air raids.

Before the War in Malaya.

Factors Common to All Troops.—The following pre-war factors in the production of neuroses in Malaya were common to all troops: (*a*) irksomeness of military training and discipline; (*b*) separation from home, with domestic ties and worries; (*c*) disruption of civilian life and career; (*d*) inability to respond fully to military modifications of civil life; (*e*) deprivation of the society of the opposite sex and of members of their own class and station in life; (*f*) pampering of trainees with easy admission to hospital, and treatment of the ill and the slightly ill in the civilian medical manner; (*g*) effects of the tropical climate, with constant humid heat, pests, parasites, skin diseases and other ailments both clinical and subclinical; (*h*) dietetic troubles and the changes necessary in a tropical climate with regard to fresh palatable foods, vegetables and fruits, as well as a possible lack of vitamin B_1, whose deficiency produces fear phenomena in dogs; (*i*) limitation of the usual sports and exercises through excessive perspiration; (*j*) a feeling of futility and waste of time experienced by many, for few in Malaya thought that Japan would be foolish enough to come into the war; (*k*) the belief of many troops that they were considered in Australia to be Malayan tourists having a wonderful time while their brothers, cousins and friends were really fighting in the Middle East.

Individual Factors.—Most pre-war subjects responded to the steel pill of Nippon and few are discussed in these notes.

One may be mentioned. He remained on at the depot and travelled with it from Tanjong Bruas, through Batu Pahat and Johore Bahru to the island and became a war neurotic. He had frequency of micturition with no discoverable somatic lesion.

A second may be mentioned. He was a complete martial misfit, boarded home during the war. Briefly, he was a French sailor, a deserter from a ship, who had not been naturalized after eleven years in Australia. He complained of head pains, tremor, and stomach pain after food. He was suffering from venereal disease and was a proven malingerer, a thief and a liar.

One more had hysteria with a fixation upon laryngitis acquired with his second typhoid inoculation in Australia. Although refractory at first, he made a good recovery as a result of strong suggestion during emergence from light general anæsthesia.

Neuroses of War.

Factors Common to All Troops.—In the ætiology of war neuroses a certain number of factors were common to all troops. The first was the inadequacy of material for training to produce efficient soldiers. This was probably due to three things: (i) the decadent conditions and easy life of civilization since the war of 1914 to 1918; (ii) the demoralizing influence of "talkies" and gangster pictures, undermining discipline and authority; (iii) the ill effects of the world depression on life, outlook and prospects, as well as the physical effects of malnutrition on adolescents and preadolescents of that period. A second consideration was the continued operation of pre-war ætiological factors in Malaya. Further training was inadequate for the type of mobile jungle warfare encountered. Physical training was not suitable, and over-motorization was a feature. At the time of onset of the neurosis medical officers were unable to provide rest, quiet and food for the patients.

There were occasional instances of lack of example to the men by non-commissioned officers and by officers. Finally, the men were engaged in active warfare, with exacerbation of the usual discomforts of army life concerning food, drink and rest, and with the ever-present element of danger to life and limb.

Individual Factors.—Among the individual factors in the ætiology of war neuroses were the following. Some subjects had had a previous nervous breakdown in civil life and required admission to hospital. Some had had a previous psychosis and had been certified insane in Australia. Others had had a previous "tropical" neurosis in Malaya. In some instances investigation of the family history revealed nervous and/or mental abnormality in parents, siblings or near relatives. Especially common was the history of a nervous mother. A number of subjects were "only" children, and one of these said that his mother was aged forty-two years at conception. A number carried on the tradition, having fathers who had suffered from "shell-shock" in the war of 1914-1918. Epilepsy was mentioned a few times in the family history. Previous head injury with unconsciousness and fractured skull was relatively common. This is sufficient ground for rejection of a recruit in the British Army. Recent somatic illness included recurrent malaria and meningitis, and one subject was convalescent from repeated ear, nose and throat operations.

Prisoner-of-War Factors.

Factors Common to All Troops.—Some factors were common to all troops. Common to prisoners in any country are the following: (i) mental depression from failure to attain the military objective; (ii) cessation of liberty of action, physical and mental; (iii) cessation of home correspondence and news; (iv) uncertainty of the future and of the fate of those at home; (v) deaths and injuries of friends and relatives in the fighting. Moreover, worry and uncertainty were present over the ultimate intentions of an unknown enemy, because of mutual misunderstandings over language, points of view and outlook on all things. There was a sudden dietary change from Occidental to Oriental type, with early rapid loss of weight and strength and the appearance of phenomena of avitaminosis. Acute rice diarrhœa occurred, then epidemic dysentery with many deaths, and then progressive alarming dietary deficiency signs. Other diseases resulted from failure of sanitation and drainage, such as malaria, diphtheria, and also cerebro-spinal meningitis and severe secondary infections in skin conditions due to malnutrition. There was gross overcrowding and hygiene was inadequate. The ordinary discomforts of tropical life were much increased. There was an acute shortage of cigarettes, tobacco and other comforts. Unit "fatigues" and manual labour were irksome on an inadequate diet. There was a shortage of general and special medicines and of vitamins, and a lack of distractions and recreations. Ration and canteen "rackets" existed. Finally, the "Selerang Square" incident revealed what our conquerors were capable of doing at any moment.

Individual Factors among Prisoners.—Dietary deficiency played a selective part in individual cases. In some early unrecognized cases of ataxic beriberi the subjects showed intense depression and labile emotions. Men suffering from cardiac beriberi were depressed and of altered temperament. Mild confusion with paramnesia was observed. Night terrors and despondency were noticed after malaria and malnutrition. Escape phenomena as hysterical fixations and conversions were seen. Homosexuality and masturbation were rare, but their occurrence was volunteered in a few cases. *Coitus interruptus* was a factor in one. Other factors were worry over the result of the war, depression and a sense of persecution, and the irritation of unit discipline, "fatigues" and food rackets.

Symptomatology of the Neuroses.

Since there was such a mixed and heterogenous ætiology symptoms presenting were legion; but all cases had one common feature, mental depression. Men suffering from war-produced neuroses displayed no euphoric traits, and the misnomer "bomb happy" was just as unfortunate an appellation as the "shell shock" of the war of 1914 to 1918.

Somatic symptoms included the following: (*a*) increased pulse rate, even rising to palpitation and to precordial pain; (*b*) increased rate of respiration; (*c*) free perspiration, pallor and flushing; (*d*) muscular weakness, even increasing to a feeling of absolute helplessness; (*e*) anorexia, nausea, vomiting and epigastric pain; (*f*) rapid loss of weight; (*g*) frequency of micturition, with glycosuria in one case; (*h*) mild hyperthyreoidism, seen only in one case.

Nervous symptoms noted were as follows: (*a*) Usually increased activity of reflexes, but not in cases of confusion. (*b*) Headache, often persistent and severe, and with different localizations. (*c*) Head "feelings", which were a frequent complaint; some of these were referred to the vertex, and one man complained of contraction and expansion "just like under an anæsthetic". (*d*) Fine tremor of the lips, tongue and fingers. (*e*) Coarse choreiform jerking affecting a whole limb or part of the body, present in severe cases. Most affected the right arm, a few the left arm, and occasionally both arms were affected together. Sometimes the head jerked with the arm and shoulder to that side. All the "jerking" cases were persistent and refractory and in all any sudden noise produced predominance of flexor contraction, with instinctive complete or partial adoption of the defensive posture. (*f*) Quavering of the voice, which was common; as the jerking of limbs varied from fine tremor to coarse choreiform movements, so did the excursions of the vocal cords vary. The voices of most showed at least a fine quaver, but in the more severe cases a gross staccato stammer was observed, so that speech was at times unintelligible. Among these patients a common feature was repetition of a word or part of a word. In the most severe acute cases the subjects simply made weird animal noises, not related to any known language. (*g*) Hysterical symptoms, which were abundant and appeared in all cases to some degree. Some of the cases were of almost pure hysteria, and among these the following were observed: (i) aphonia, which was fairly common; (ii) pareses of the skeletal muscles, both as fixations and as conversions; (iii) topical preoccupations, mostly referred to the head or to the stomach; reference to the heart was rare.

The psychic symptoms were as follows. Affectively, all subjects showed depression, while labile emotions, emotive facial irritability and change of temperament were noticed. Conative activities and inactivities were observed in panic and paralysis fear reactions and reaction tendencies; combination of both in one subject was seen.

Cognition in its reaction responses presented the most mixed and variegated features. (i) Amnesia varied from partial to total. Few subjects had any clear memory of details of the first few hours succeeding their neurosis-producing experience, while one could remember nothing of his life or even his name. His most recent memory was of the tall building (the Cathay Theatre) in Singapore subsequent to the capitulation. (ii) Insomnia was common and was due to reliving the incapacitating incident, and if sleep came, to night terrors regarding the incident. (iii) Mental confusion was common early in the neurosis. (iv) Ideation was slow and retarded early, and went *pari passu* with the mental confusion. (v) Loss of powers of attention were due to loss of power to concentrate, to easy distractibility to ever-present fear and to the recall of recent experiences. (vi) Sensitivity to noise was pronounced and was probably the most persistent feature in all cases of neurosis. Some of these cognitive derangements were common to many cases; but in certain subjects one or other feature predominated, and this imparted to the total picture of the neuroses gross apparent dissimilarities.

Treatment of the Neuroses.

During the pre-war period the Second/Second Australian Convalescent Depot was ideally situated for its purpose, including the treatment of men suffering from neurosis. Unfortunately the Japanese swept down the western side of the Malay Peninsula, leapfrogging down its coastline,

and the depot both at Tanjong Bruas and at Batu Pahat was in their path. When all troops had been withdrawn to Singapore island there was no place free from noise or immune from danger. Consequently at no time during the war were conditions suitable for the treatment or rehabilitation of neurosis patients.

Nevertheless, treatment was undertaken and some success was achieved. Bromides, "Luminal", "Soneryl" and occasionally morphine were used to secure active sedation. After a few days of this patients were given graduated light exercises in the sun and fresh air. This was combined with individual full explanation of the condition, with sympathy in judicious doses, and with reassurance regarding the patient's powers of self-control and endurance. Quiet but insistent explanation and reassurance did most in restoring the patient's loss of confidence in himself.

Occupational therapy of a somewhat industrial character was employed during the war. Patients were required to dig their own slit trenches, erect their own tents, secure water, provide for sanitation *et cetera*. The results were good, but on occasion, and especially after a near air raid, fairly strong "pep" talks were required. After capitulation this form of therapy had greater scope, and one patient may be mentioned; employed as bookbinder at the education centre, he completely regained all confidence in himself. He subsequently did a splendid job in Japan in very adverse circumstances, and perished off Formosa on the way home.

Exceptional cases required exceptional treatment. Strong suggestion on emergence from light general anæsthesia succeeded in a case of aphonia, but failed in a case of skeletal paresis. Appreciation must be expressed of the full prophylactic significance of the many amenities provided at Changi prisoner-of-war camp by our own authorities and by the high-minded and unselfish activities of artists, musicians, lecturers, organizers and sportsmen. A prophylactic factor of paramount importance was the early inculcation of rigid discipline by our camp commander, known to all Allied prisoners at Changi as "Blackjack".

Results Obtained in the Neuroses.

Neuroses Present Before the War in Malaya.

Most pre-war neurotics had been eradicated by medical boards and repatriated by the time the war in Malaya began. The residue vanished with the outbreak of war.

Neuroses Occurring During the War.

Among war-time neurotics, most came to the convalescent depot and received treatment. Some were returned to their units and to action. The remainder, with few exceptions, were cured immediately upon the capitulation and the removal of danger stimuli. A residue of four severely affected patients, each with gross jerking and stammer, remained with the depot. They required six months for their complete rehabilitation.

Neuroses of Prisoner Life.

Prisoner-of-war patients received individual treatment and in trying circumstances did well, with the exception of two suffering from hysteria, who failed to respond.

Conclusions.

Neuropaths and psychopaths are potential encumbrances, and though just as patriotic as other volunteers, they should have their condition explained to them as soon as it is detected at enlistment, at attestation or in training camp. They should never leave Australia on any form of active service, and they can all be usefully employed at home. Greater care, foresight and attention are required at attestation with regard to mental condition. Frequent delinquents, chronic sick parade attenders, draft-dodgers and those suspected of being neurotic should undergo psychiatric examination in training camps. Potential neurotics, in whom the neurosis will be precipitated by war conditions, can be detected during transport. Psychiatry has not yet received full military appreciation.

PART II: PSYCHOSES.

Before the War in Malaya.

No pre-war psychotics were encountered by me in Malaya, though one was discovered at Redbank Camp, Queensland, prior to embarkation. This subject claimed to be both Napoleon Bonaparte and Jesus Christ. He admitted having been previously certified six times and admitted to the Brisbane Mental Hospital. Cases of psychosis must have occurred in Malaya before the war, but I have no knowledge of them.

During the War.

During the war one case only of psychosis was encountered.

This subject came to the convalescent depot during its short stay at Batu Pahat from the Tenth Australian General Hospital, which was about to be evacuated from Malacca to the island. During the ambulance trip of some 70 miles the convoy had to hide twice from marauding Nipponese bombers, and the patient, a recovered venereal disease subject, broke down and required restraint. He heard the voices of his doctor at Malacca, and of his wife and child in Sydney, both of whom he had murdered "with the pox". Red-eyed, agitated and depressed, he demanded a gun, sword or bayonet, in order that he might destroy himself. He was not detained by us, but went straight on to the Thirteenth Australian General Hospital, which was then occupying part of the Johore Mental Hospital at Tampoi. He was seen only for a few minutes and then not again.

Psychoses of Prisoner-of-War Life.

Psychoses of prisoner-of-war life were encountered at Selerang and at the Roberts Hospital, Changi. This hospital contained some 2,500 beds, and its mental ward catered for all Allied psychotic patients. British, Australian and Dutch were treated, the last category including white or Holland Dutch and Chino-Malay Javanese Dutch. Only cases among the Australians are described; but a short synopsis of all cases is appended. Two very severe cases of neurosis of war production verged upon psychosis, but the subjects responded to therapy.

Reports of Cases.

The following are the cases of certifiable mental illness that occurred in the first fifteen months of prisoner life among a population of some 15,000 Australians, 35,000 British and an unknown number of thousands of Dutch prisoners.

CASE I.—The patient was aged thirty-eight years, and the condition was of sudden onset after he had been a prisoner for two months. He had had previous mental trouble in 1934 and at Bathurst camp in 1940. He was the thin, agile type with a kyphotic spine, and was previously a professional dancer, addicted to alcohol and to low women. A scar across his throat suggested attempted suicide; nothing pertinent to his mental illness was discoverable at physical examination. The patient was admitted to hospital with visceral and nihilistic delusions, labile emotions and apparent apathy; he showed hyperacusis and hyperæsthesia; his habits were variable but mostly clean. Psychomotor activity was variable; at times complete immobility was present.

After being in the ward for eight months he suddenly became mentally lucid, gave a good account of himself and behaved normally for two days. Then suddenly progressive heart block developed and he became comatose and died. Post-mortem examination revealed tertiary syphilis of the thoracic part of the aorta and its arch and of the occipital bone, bilateral pachymeningitis and atrophy of the frontal convolutions.

This case was one of a manic-depressive reaction type of psychosis with syphilis superimposed.

CASE II.—The patient was aged twenty-seven years. The onset of the condition was rapid after he had been a prisoner for three months. He had not had illness of any kind prior to coming to Malaya, and his family history was clear of mental or nervous taint. As a prisoner he suffered from recurrent malaria, dysentery, and rapid loss of weight and strength from malnutrition. The man was of splendid physique and a good athlete and footballer. Clinical examination revealed nothing relative to his psychosis. On his admission to hospital he displayed increased psychomotor activity and lability of emotions (mainly depressive) and

had auditory hallucinations. From his psychotic phase he rapidly reverted into a syntonic personality type. He was rehabilitated after six months and returned fit to his unit.

This was a manic-depressive reaction type of psychosis precipitated by illness, malnutrition and prisoner-of-war depression.

CASE III.—The patient was aged twenty-five years. The onset of the condition was after he had been a prisoner for three months. He had previously been healthy and his family history was good. The onset was gradual, when he was weak from recurrent mild fever and malnutrition. He was restless, uttered nonsense phrases, and postured, and was affected by echolalia, confusion, loss of memory and a depressive mental background with regression to a childish mode of speech and choice of words. In the early stages of his illness he displayed evidence of cerebral avitaminosis with pseudomeningitis, which cleared up on the exhibition of "Marmite". He had not received active or suppressive malarial therapy since his admission to hospital. During a bout of pyrexia, without other symptoms, his blood was examined and a heavy *Plasmodium vivax* infection was found. It was noted that with the fever he became mentally lucid and that he recovered his memory; furthermore, he gave a good detailed history of himself and his family. He told us that he had had repeated attacks of mild untreated malaria since his unit had been at Mersing the previous year. The present attack, during which he maintained his clarity of thought, was deliberately untreated and allowed to die out spontaneously; but a daily watch was kept upon a thick blood smear. After a week free of fever, during which he did not regress mentally, fever again developed, and this was vigorously treated and succeeded by suppressive therapy. He made rapid progress towards rehabilitation and was returned fit to his unit.

This patient was a schizophrenic showing palæophrenia caused and relieved by malaria. He was followed up for three months after his discharge from the mental ward, and he still maintained his recovered normal mentality.

CASE IV.—The patient was aged twenty years, and his condition came on gradually in a medical ward after six months' prisoner life. He had previously been healthy and had a good family history, and he early suffered from severe dysentery followed by cardiac beriberi. While he was still in a medical ward suffering from the latter condition he became strange in manner, noisy and restless, and was transferred to the mental ward. Here he had auditory hallucinations and made antics, postured, laughed and smiled without apparent cause. He was apathetic to questions about his family, a most unusual thing among prisoners of war. He exhibited fixation upon his cardiac beriberi by sending and receiving messages through his heart by means of a "cardiogram". Of a pleasant nature, he was clean and tidy in his habits, and although he had no insight into his mental condition he was orientated for space and time and was aware of his immediate environment and of the identity of his fellow patients. Schizophrenia existed in a pure state, for when he was spoken to quietly in the midst of his muttering over messages through his "cardiogram" he would immediately smile, greet me and answer the question asked, again to resume his telegraphy operation. He was given extra food and "Marmite" received through the British Diet Centre. His rehabilitation was sufficient after six months for him to return fit to his unit and, observed for a further two months, he remained quite well.

This case was one of schizophrenia arising out of malnutrition and cardiac beriberi; it was probably constitutional and only precipitated by these factors.

CASE V.—The patient was aged twenty-two years, and his psychosis developed gradually after four months of prison life. He was observed in his unit in Australia to be solitary and mysophobic and to laugh and smile with no apparent cause; he came to Malaya, where he served no useful purpose in the war. During most of that period he was a unit-retained war neurotic. His physical state remained normal during his early prisoner existence, but after four months he was adjudged to be insane and was sent to the mental ward. He gave no trouble; he was clean and tidy, but was totally devoid of initiative and completely apathetic towards other patients and to questions regarding his own family. He laughed and smiled to himself and was always solitary. He never revealed his mental content. Slowly he responded to visits from fellows of his unit and to day visits over to Selerang, and finally he expressed a wish to be back with his unit. He was discharged and was examined three months later, when he was doing simple, useful jobs at his unit headquarters; he was still a simple, harmless schizophrenic.

This patient was a simple schizophrenic who should never have left Australia; ample evidence of his mental state in the training camp was provided by officers of the unit.

CASE VI.—The patient was aged twenty-two years. The onset of his psychosis was gradual, after four months of prisoner life. He said that he had previously had good health and that his family history was one of perfect harmony and happiness and devoid of mental disease. Prior to capitulation, with a special party of 44 in a motor-boat he left Singapore. They were forced ashore on Jibbia Island by the enemy and their boat was disabled. They had little food or quinine, and they were marooned for three months before the Nipponese came back for them. In that time more than half the party had died, and the patient suffered from daily mild attacks of malaria. He was taken to a civil jail in Singapore, and from there came to Changi. In a medical ward he had early beriberi, severe anæmia, malaria and a palpable spleen. While in the medical ward he became depressed and began to hear accusatory voices. After hearing these for two weeks he decided to end his life, and he severed his left radial artery with a safety razor blade. After his admission to the mental ward he was consistently depressed, yet perfectly orientated for time and space, clean and tidy, quiet, and not in any way troublesome, eating and sleeping well and maintaining fair bodily health. He showed no initiative and would not voluntarily do any ward work or join in card games or amusements. He did so when asked to, but seemed to derive no pleasure from them. He answered all questions freely and without reserve, but he persisted in the opinion that he could not go home in the condition he was in and that suicide was the only way out. The voices did not worry him as they used to, but they were still present. He had insight and knew that he was mentally ill.

This case was one of depressive psychosis due to malaria.

CASE VII.—The patient was aged thirty-eight years. His psychosis was of sudden onset after two months of prisoner life. He was normally strong and healthy and his family history was clear. He contracted malaria during the war and was treated for it. Early in his prisoner-of-war life he suffered from dysentery, from which he recovered. It was some months after his last malarial attack or treatment when he became psychotic. He was restless, noisy and agitated, and exhibited visceral and persecutory delusions. This state was followed by a period of semistupor with retardation and melancholia. On occasion he complained of low right-sided pain; he also said that he had lost his sense of smell and of taste, and that he had not passed urine or fæces for weeks.

Examination revealed no surgical condition; his urinary and defæcatory functions were normal, but he had a low fever. Examination of his blood showed a heavy *Plasmodium vivax* infection. Vigorous antimalarial treatment was instituted, and his response was good. He rapidly regained mental clarity and body weight. He was discharged fit to his unit.

This case was one of a depressive psychotic state with cyclothymic aspect caused by malaria. His visceral delusions had a somatic foundation and took the form of a fixation upon his dysentery. Immediately upon the clearing of his dysenteric state he went eleven days without a motion and he had low right-sided pain during that period.

CASE VIII.—The patient was aged thirty years and the onset of his psychosis was gradual after four months of prisoner-of-war life. In a happy and clear family life he was frustrated in his ambition to be a priest through lack of scholarship. He made vows of celibacy to himself and maintained them. He had a stormy adolescence and young manhood and fell in love with a girl at his place of work. He never declared his love; but the girl was the cause of frequent nocturnal emissions and of his masturbation. He sought medical advice, but it was without effect. Although he was fond of games and active and strong, he still persisted in masturbation. As a prisoner he suffered early from dietary deficiency, rapidly lost weight and strength, and had scrotal dermatitis, stomatitis and painful feet. He slept badly, and after two months he began to think that his mates were talking about him, especially when he came back from the urinal during the night. He thought they were laughing and saying that he had been masturbating again. When tackled by him they denied it. He soon lost all his mates and was involved in many fights. Finally he was sent to the mental ward. Here he was found to be perfectly normal, except for his ideas of reference and actual accusatory hallucinations, which always had the same content. He had read psychology voraciously for years, and he admitted

the possibility of being hallucinated; but he persisted in his beliefs. He could be said to have insight into his lack of insight. In the mental ward he showed no improvement except physical improvement. Fever and high right-sided tenderness developed, and on his transference to a medical ward amœbic hepatitis was diagnosed.

The obstinacy and simplicity of his mental state suggest paranoia. His removal to a medical ward coincided with my departure to Japan. This would be an interesting case to follow up.

CASE IX.—The patient was aged twenty-nine years, and his psychosis had its onset after seven months of prisoner life. Unit complaints of persistent malingering delinquencies led to his admission to hospital for observation. He was found to be suffering from simple schizophrenia of lifelong duration, his condition being mildly aggravated by the depressive conditions of life as a prisoner of war. He responded well to the environment of the mental ward. He was returned to his unit, with explanations to the authorities of his unit for his behaviour tendencies, and he did not return to us.

This patient was a simple schizophrenic, harmless and well capable of some useful work in his unit.

CASE X.—The patient was aged forty-two years. His psychosis had its onset after seven months of prisoner life. He had previously been healthy. He admitted being illegitimate and denied having had any previous mental or nervous trouble. He was first examined by me when he had a prisoner-of-war neurosis; he was referred for periodic attacks of depression and crying preceded by irritability, and responded to psychotherapy. Later, when he was again at the convalescent depot, recovering from amœbic dysentery, he displayed fits of ungovernable temper. These were always preceded by ideas of reference, from which he quickly worked up first to an argument with some inoffensive person and then to a bodily attack upon him. From the time of his admission to the mental ward he entered upon a docile, willing and obedient form of behaviour, and he seemed well conversant with the routine of mental treatment. A suitable job was found for him and he was returned to his unit.

Tests for syphilis gave negative results, and only his rages suggested epilepsy. It seems probable that he was suffering from a depressive psychosis, constitutional and precipitated by prisoner-of-war conditions of life. The psychosis had a strong syntonic suggestion.

CASE XI.—This patient was aged thirty-eight years. The onset of his condition was gradual and prior to his becoming a prisoner of war. The history of his previous life was normal and his family was free of mental taint. His commanding officer informed me that during the last few days of the war the patient's manner underwent a change. He had expressed worry for some time about not receiving letters like the other men did, and admitted that he had left Australia "under a cloud", having quarrelled with his wife. He became irresponsible after being trustworthy, and did strange things. As a prisoner he rapidly became worse and physically he failed rapidly. He was referred to me, and consultation revealed that he was dirty, untidy and excessively euphoric. He was assertive and boastful, and claimed prodigious powers of body and mind. He could speak all languages and he knew all medicine. His father was also a doctor and the head of the secret service in Malaya. He had taken the Prince of Wales around the world in a yacht, and so on. He decked himself with gauds and trifles. Clang association was marked and distractibility was easy. A harmless euhphoric good nature prevailed, and he was allowed to stay with his unit, all personnel of which agreed to look after him. Going his own easy way through prisoner life, he soon developed a prodigious appetite and began to put on weight rapidly. With increase of weight he regained his normal mental state, becoming clean, tidy, responsible and industrious.

This was a true manic-depressive reaction type psychosis precipitated by domestic worry and separation and aggravated by the conditions of early prisoner-of-war life.

Two further cases are worthy of mention; both these psychoses were terminal phases of a somatic condition. The first was an early case of gross dietary deficiency prior to the recognition of pathological states due to avitaminosis.

This man simply could not tolerate rice, and he wasted away for six weeks in his unit camp hospital. A small quantity of extra foods came to the convalescent depot, and he was sent to us to be fed up. On arrival he was literally skin and bone. Mentally, he was clear, pleasant and good-natured. Suddenly, on the second day at lunch-time, an orderly reported to me that the patient was behaving strangely. He was found laughing and smiling to himself, and he told me as he broke cigarettes up into his "vegetable" soup that he was putting cream on the lovely strawberries that those two (non-existent) young men by his bed had just given him. An earlier decision of that day, that he go to hospital on the afternoon ambulance, was hastened, and he was transferred immediately. From hospital reports it was learned that he arrived there in a state of fever, semicoma, facial twitching and muttering delirium. He died in a few days. His cerebro-spinal fluid was opalescent and contained 40 cells per millilitre, all cells being lymphocytes. Post-mortem examination revealed a pink condition of the cerebrum and acute encephalitis with œdema and congested blood vessels.

To what extent were the mental signs due to avitaminosis or starvation? Was the condition purely a terminal infective state? Did Werneke's syndrome enter into the question? At this time vitamin deficiency signs were not generally recognized and there had been no deaths from cardiac beriberi or ataxic patients with mental signs.

The second of these two cases occurred in Japan.

The patient was a member of "J" force from Changi. Aged forty-eight years, he had suffered from cardiac beriberi at Selerang, and since his arrival in Japan he had worked hard and well. He was a tower of moral support to his younger workmates. His unfailing good humour and spirits helped many to carry on. Early in our first winter he suffered from a heavy cold, then severe influenza. It was not possible to obtain permission for him to stay in camp away from work, and he was admitted to hospital with obvious pneumonia. He seemed to be about to recover, but on the fourth day he relapsed and passed from delirium into a state of acute confusional insanity. He was euphoric and completely disorientated for time and space. The phase lasted for two days, and he died quietly of exhaustion and heart failure.

***Findings.*—The findings in these cases of psychosis may be summarized as follows. Eleven psychoses occurred among 15,000 prisoners. The average age of the patients was thirty-three years, and the average time of onset was after four months of life as a prisoner of war. The diagnoses were as follows: manic-depressive psychosis, three cases; depressive reactive psychosis, three cases; schizophrenia, four cases; paranoia, one case. The ætiology was as follows: precipitated by prisoner-of-war conditions, five cases; due to dietary deficiency, three cases; due to malaria, three cases. The results obtained were: recovered and returned to unit, eight patients; mental condition unchanged, improved physically, two patients; died, one patient.**

***Treatment.*—Special treatment, such as insulin, "Cardiazol" *et cetera*, was not available. Extra food and "Marmite" were given to all whenever these were able to be obtained. The mental ward was fed by a British kitchen, and the British Diet Centre gave all it could to mental patients, irrespective of nationality. Social rehabilitation was our only standby. Every effort was made by a good staff to treat all patients as though they were normal persons. They were kept clean and tidy, shaved and bathed. All were encouraged to join in card games and other amusements. Visits by friends from units at Selerang were organized. Excursions of patients to units were similarly organized. Visits to sports fixtures and concerts were encouraged. Any social improvement in a patient was made an occasion of sincere congratulation to him. Similarly, spontaneous efforts of patients to help in the ward were encouraged. The results obtained by these methods of intimate contact with the life and progress of each patient, when we were denied the use of modern forms of treatment, have convinced me that intensive social rehabilitation is of greater importance in obtaining a remission than shock therapy. The latter at best can only be a preliminary adjunct to treatment. Case III is of particular interest. It would appear that undiscovered malaria was largely responsible for this patient's psychosis. Further, it would appear that a more than usually severe attack, possibly a new infection, was responsible for his recovery of normal mental faculties.**

Survey of All Cases of Psychosis at Changi.

The following is a brief survey of all patients admitted to the mental ward, Changi.

There were four Dutch patients. Their histories are summarized as follows.

CASE XII.—A white patient, aged eighteen years, suffered from acute mild schizophrenia of sudden onset coincident with acute appendicitis after being a prisoner for six months. He became normal immediately upon the removal of his appendix (which had a gangrenous tip) and remained so under observation for a further six months.

CASE XIII.—A Chino-Malay patient, aged twenty-two years, had a depressive psychosis of gradual onset. He gradually recovered with extra food and "Marmite".

CASE XIV.—A white patient, aged thirty-eight years, had a depressive syntonic psychosis of gradual onset. He gradually recovered with extra food and "Marmite".

CASE XV.—A Chino-Malay, aged forty-one years, suffered from severe peripheral and central malnutrition neuritis, with ataxia and spasticity and a depressive confusional psychosis. Extra food and "Marmite" relieved the mental state, but produced no change in his somatic condition.

Eighteen patients were British. A summary of their histories follows.

CASE XVI.—This patient, aged thirty-eight years, was suffering from a depressive confusional psychosis, which was cured with "Marmite".

CASE XVII.—This patient, aged thirty years, arrived from England in January, 1942, suffering from schizophrenia.

CASE XVIII.—This patient, aged thirty years, was suffering from a severe suicidal neurosis of dietary deficiency. He was cured.

CASE XIX.—A patient, aged twenty-nine years, was suffering from catatonic schizophrenia precipitated by prisoner conditions, and from malnutrition. His condition was relieved by extra food and "Marmite".

CASE XX.—A patient, aged twenty-seven years, had a mild depressive psychosis which was cured by extra food and "Marmite".

CASE XXI.—This patient, aged twenty-five years, suffered from episodal amnesia. His was a compassionate admission to hospital to avert the wrath of the Japanese and punishment.

CASE XXII.—A patient, aged twenty years, referred as insane, was found to be illiterate and a victim of unjust military punishment. He was retained in the mental ward and taught to read and write.

CASE XXIII.—A patient, aged twenty-five years, suffered from stuporose catatonia; his condition remained unchanged throughout.

CASE XXIV.—This patient, aged twenty-six years, was suffering from hebephrenic schizophrenia; his condition remained unchanged throughout.

CASE XXV.—A patient, aged thirty-five years, suffered from a depressive confusional psychosis; he was cured with "Marmite".

CASE XXVI.—A patient, aged twenty-five years, suffering from a depressive psychosis and obvious pellagra, was cured.

CASE XXVII.—A patient, aged twenty-six years, suffering from hebephrenic schizophrenia, was relieved.

CASE XXVIII.—This patient, aged forty-five years, was suffering from emotional syncope (anger); he was retained to avoid punishment.

CASE XXIX.—This patient, aged twenty-six years, suffered from simple schizophrenia precipitated by prisoner-of-war conditions. His condition was relieved.

CASE XXX.—A patient, aged twenty-five years, suffered from simple schizophrenia; he was relieved by "Marmite".

CASE XXXI.—This patient, aged twenty-three years, suffered from the depressive psychosis of chronic dysentery; his condition was noted to be improving.

CASE XXXII.—This patient, aged forty-four years, a Hong Kong prisoner, had a manic-depressive psychosis, of gradual onset, in Japan; the condition was very refractory.

CASE XXXIII.—This patient, aged twenty-eight years, was a religious homosexual and suffered from mild mania. His condition remained unchanged.

One patient was an American.

CASE XXXIV.—This was a case of schizophrenia in a patient, aged twenty-eight years, precipitated by depression of prisoner-of-war life. He made two determined attempts at suicide, once by jumping into the harbour (he contracted pneumonia) and once through a small window. His condition had been improved to almost complete remission when the war ended.

In addition to these cases, one Englishman and one Australian were prompted to feign insanity and were coached in the part. Each had attempted escape and each had been caught. They received sanctuary in the mental ward and were not detected on examination by Nipponese doctors. After some months, when the excitement had died down, they were quietly drafted into work parties upcountry and thus escaped execution by shooting.

Summary.

The information about the total number of 34 cases recorded, amongst all nationalities, may be summarized as follows. The average age of the patients was 29·5 years. The following diagnoses were made: schizophrenia (average age twenty-six years), 14 cases; manic-depressive psychosis (average age thirty-six years), seven cases; depressive-reactive psychosis (average age thirty years), 10 cases; no psychosis, three cases. The ætiology was recorded as follows: constitutional causes, 17 cases (six patients were relieved by "Marmite"); malnutrition, nine cases (three patients were obviously suffering from skin pellagra); appendicitis (cured by operation), one case; malaria (undoubtedly connected with the psychosis), four cases; three patients were not psychotic. The following results were obtained: 23 patients were relieved and discharged to their units; seven remained unchanged mentally; one patient died; three patients were not psychotic.

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SOME PROBLEMS OF BACKACHE AND SCIATICA.[1]

By LEONARD LINDON, M.S., F.R.C.S.,
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THE intervertebral disk has of recent years become headline news in our journals, and the controversy of opinion between the two sides of our continent reminds one of Kipling's "Ballad of East and West". So much has been written on this subject, so much of wishful thinking, so much of pious opinion unsupported by anatomical fact, so much of optimism unfulfilled, that I am diffident of putting before you conclusions drawn from my meagre series. But of one thing I am certain—a sound operative procedure is in danger of being grossly abused. There is not enough thought for the future, based on pathology. And the first question the patient and his physician are entitled to ask is this: "Just how necessary is surgical exploration in this particular case?" From a series admittedly very small, I am going to try to answer that question.

It has frequently been stated that the vast majority of cases of low-back pain and sciatica are due to injury or disease of the intervertebral disk, and with this statement in general one must agree, if we use the words "associated with", rather than "due to". But the problem of diagnosis is not so simple as that. I am mainly concerned in this paper with the diagnosis of cases of so-called sciatica. There is no difficulty in the neuroanatomical diagnosis of root involvement, though there may be uncertainty as to which root is involved, or as to how many roots are affected. But the diagnosis of the actual pathological condition present is not nearly so easy; in my small series of 72 explorations, the condition predicted prior to operation was found to be present in only 48 cases, and this percentage agrees closely with the findings of certain conservative

[1] Read at a meeting of the South Australian Branch of the British Medical Association on May 2, 1946.

and entirely reliable American observers—for example, F. C. Grant. That is to say, in only 48 of 72 cases was the condition of a "ripe" extrusion of the *nucleus pulposus* found. I shall explain later what is meant by the term "ripe" extrusion. It is obvious that the most difficult problem is to give a pathological diagnosis which will account for the clinical condition present in each case, and it is at this point that weakness is apparent in the statements of those who blame the diseased disk for all low backache and root pains, and claim to cure all such pains by removal of the disk—if such a thing was possible, which it is not.

I shall attempt to describe various conditions found to be present during the course of 72 operations performed with wide exposure in the exploration of disks and nerve roots at the lumbo-sacral junction, and to see how far the interpretation of symptoms and signs is confirmed by operative findings. This is admittedly a small series, but it provides adequate material for analysis and criticism.[1]

The following is the probable life history of a degenerating disk: (i) repeated minor traumata occur; (ii) the *annulus fibrosus* is weakened and the *nucleus pulposus* loosened; (iii) either (*a*) a more severe injury then ruptures the annulus, and the nucleus is extruded (sequence of back pain followed by root pain—spontaneous absorption may follow), or (*b*) steady degeneration and absorption of the disk continue, the disk space is almost obliterated, the relation of the facets of the apophyseal joints is altered, and chronic traumatic arthritic or osteo-arthritic changes follow; these arthritic changes may not be productive of symptoms, until a specific gross injury or strain produces an exacerbation of the lesion; spontaneous fixation may follow. This last-mentioned condition—that of arthritic changes or abnormal mobility in the apophyseal joints—has been emphasized by Dr. N. S. Gunning. The apophyseal joints are supplied by branches of the posterior primary divisions of the appropriate nerves, and it is found in many other parts of the body that if the articular branches of a nerve root are inflamed or irritated, their pain may be referred to the cutaneous area supplied by that root, and to the muscles supplied by that root. Such muscles will undergo wasting. I mention this to emphasize that root pain does not necessarily mean a mechanical involvement of the nerve root; in fact, in this small series, in one-third of the cases there was no evidence of root compression.

One reads also of low back pain being due to disk trauma. It is doubtful whether an intervertebral disk of cartilage has any pain sensibility at all. I am certain that most of us can remember occasions during lumbar puncture when the disk has been penetrated by the needle, without the production of pain. But there is no doubt about the pain sensibility of apophyseal joints, or of any other joint which possesses a synovial membrane.

It is best to base one's conclusions on actual operative findings, though occasionally one must make deductions based upon pathological analogies.

Definite Operative Findings.

The "Ripe" Nucleus Pulposus.

By the term "'ripe' *nucleus pulposus*" I refer to the lesion most obvious and decisive at operation, and the one most dramatic in its relief. At exploration, removal of half the lamina of the fifth lumbar vertebra reveals the fact that the first sacral root is grossly compressed and absolutely fixed and taut. It is tightly stretched over a mass of nuclear tissue, which is projecting backwards from the lateral aspect of the disk, generally in the vicinity of the apophyseal joint. The nerve is tense and flattened, and in certain cases the nerve is also nipped posteriorly against the lower articular process of the particular apophyseal joint. In such a case the pain is really intense. This condition of the nerve is in the most pronounced contrast to that of the nerve on the unaffected side, which is loose and mobile. Such a case is suggested clinically by the signs of gross root involvement, and by pain which is intense, incessant and not relieved by prolonged rest or immobilization. Often acute hyperæsthesia of the appropriate area of skin is present. This almost certainly represents a late stage in disk disease; the nuclear material has been loosened by repeated small strains, productive of no serious symptoms except occasional low-back pain—minor lumbago for which the patient does not seek relief. Many of us over the age of thirty years have such attacks of lumbago. And then some final strain ruptures the lateral portion of the annulus, and the nuclear tissue is extruded. At operation, if the nerve root is retracted—and this is often difficult to do without increasing the injury to the root—the nuclear tissue is found to be projecting like a small green pea in size. If the capsule over it is incised, the nuclear tissue is extruded in one piece under its own pressure. For the sake of brevity, I refer to this as a "ripe" *nucleus pulposus*. The immediate relief is dramatic. The late results will be referred to later.

Organization of an Extruded Nucleus Pulposus.

A few patients, with longer histories and suffering from intermittent attacks, have had what I believe to be organization of an extruded *nucleus pulposus*. A mass of nuclear tissue, easily recognized, is found lying within the neural canal, adherent to body in front and to nerve root or roots behind, and organization and absorption are taking place. In response to this reaction, one finds the epidural fat tough and infiltrated, and epidural hæmorrhage often troublesome. One cannot but think that these changes represent what happened in cases of intense sciatica in days gone by, in which a long and painful malady was followed by spontaneous and complete relief of pain.

Calcification in a Diseased Disk.

Calcification in a diseased disk is a late change, found in patients who have a long history of fairly constant backache with interludes of severe sciatica. I believe that in nearly all these cases the apophyseal joints are the site of changes—call them traumatic osteoarthritis—which are responsible for the local pain. The sciatic root pain is produced when the nerve is stretched over the calcified projection. Why, then, is intermittent pain so common a feature in these cases? I think one must postulate that for some reason unknown there occurs some swelling or œdema of the epidural fat and connective tissue, enough to interfere with the mobility of the nerve root in relation to the disk projection; this would probably cause œdema of the nerve and its sheath, with ensuing pain and loss of function. This is not entirely a fairy-tale. On five occasions I have found the suspected nerve root to be swollen and œdematous, and its sheath intensely vascular. This suggestion offers explanation of the fact that so many cases of sciatica have followed chill and exposure to cold; this has been a particularly common story amongst servicemen.

Doubtful Operative Findings.

Hypertrophy of the Ligamentum Flavum.

The finding of hypertrophy of the *ligamentum flavum* has frequently been recorded by observers, but it lacks conviction, because it is difficult to know what constitutes the normal size of the ligament. However, in this series, several patients with clinical evidence of root involvement were subjected to operation, and the only abnormality that was found was a massive ligament, particularly the interarticular ligament; also the epidural fat appeared to be in excess, and the extradural tissues were unusually vascular. Although the explorations were regarded as giving essentially negative results, the patients all experienced great relief, but not such dramatic relief as follows the removal of a "ripe" nucleus. May not infective swelling and œdema of soft tissues be factors in the production of root involvement in such cases?

The Concealed Disk and the Mobility Test.

The concealed disk and the mobility test are, I believe, good examples of wishful thinking. It is difficult to believe

[1] Various operative findings and deductions were illustrated during the course of the paper by X-ray films and diagrams.

that the alleged concealed disk can have any part in the production of root pain, or that an assault on such a disk has any justification. With regard to the question of the mobility test, I feel certain that many of you have been present during laminectomy for suspected tumour of the cord when the degree of anæsthesia has unhappily been allowed to grow too light, and the patient has strained with forcible expiratory effort. The result is a good exhibition of the normal mobility during flexion of the spine. It seems to me that the so-called mobility test was coined by chiropractors.

Treatment.

When one is endeavouring to decide the appropriate operative treatment for any patient suffering from backache and sciatica who has failed to respond to conservative measures, the most important problem is to determine whether the apophyseal joints have become involved, and what is the degree of that involvement. In my experience, involvement of these joints may be expected when the following conditions are combined: (i) long duration of the condition; (ii) a patient following an energetic occupation; (iii) a patient aged over forty years. Such involvement can generally be diagnosed on the history, on the symptoms such as occur in chronic traumatic or degenerative arthritis in any joint (stiffness and pain after rest, relief by immobilization and by the application of heat), and by local signs (poker back *et cetera*) and involvement of the posterior primary divisions of nerve roots. X-ray pictures may help, but the diagnosis is essentially clinical. In brief, surgical treatment may be summarized as follows:

1. If the diagnosis of intense and persistent sciatica due to the extrusion of a *nucleus pulposus* is made, the operation of removal of the nuclear tissue gives dramatic relief, shortens the convalescence very considerably, and (as far as we know up to date) gives a good prognosis for return to most occupations. But it would be foolish to claim that a perfectly normal back results.

2. In other cases, in which apophyseal joint involvement is suspected or certain, the choice lies between conservative measures and some type of fusion operation. Such an operation may not result in the patient's return to heavy labour, but he will be able to do useful work, and will enjoy relief from his disabling pain.

Results.

Finally, let me give a brief summary of results. I have on three occasions had to perform a reexploration for recurrent sciatica in patients who had had their first operation performed in army hospitals, and I believe that in each case the fault lay, not in the operative technique, but in the wholly inadequate period of convalescence described. I may be too conservative; but it is my practice to keep such patients in bed for three weeks after operation. Physiotherapy in bed can be commenced two days after operation, and patients are encouraged to perform their own exercises in bed. Thus, when they are allowed up, they do not experience either pain in the leg or pain in the wound, and their subsequent recovery of economic function is all the more rapid and complete. These patients have all experienced prolonged and exhausting pain prior to operation, and on the psychological side alone, any post-operative recurrent pain has a most demoralizing effect. The three patients to whom I have referred had all been allowed up too soon, and had been pursuaded to attempt too much too soon. Those patients who followed moderately heavy occupations have been able to return to them. But I am of the opinion that it is wrong to expect that in any of these cases a perfect result is achieved, and that the patient is fit for the heaviest work.

Conclusion.

Keegan states that the operative treatment of this type of sciatica merely cuts short the course of the disease, and that it must leave the back a little less normal than prior to the onset of the condition. It seems to me that he expresses a fair and honest summary, and one which will be abundantly confirmed with the passage of time.

Reports of Cases.

A MIXED TUMOUR OF THE PALATE WITH SARCOMATOUS CHANGES.

By E. A. JOSKE,
Balaklava, South Australia.

Clinical Record.

IN February, 1946, Mrs. C., aged fifty-one years, presented herself on account of increasing difficulty in swallowing. The pertinent features of her past history were as follows. The tonsils had been removed some ten years previously. She had sought advice four years earlier because her throat was "uncomfortable", and was assured no ailment requiring treatment was present. Examination revealed a defined swelling of the right side of the neck, covered in part by the mandible, and about the size of a walnut. It was not determined whether this was a gland or whether it was part of a tumour which extended medially to the mandible and laterally to the old tonsillar bed, to occupy the soft palate. Pressure on the right side of the neck caused a corresponding movement through the tumour into the mouth and palate. The passage to the pharynx was reduced to about half its normal size. Examination down to the vocal cords showed normal structures. X-ray examination failed to reveal the shadow of a salivary gland calculus.

A diagnosis of mixed tumour of the palate was made. The likelihood of malignant change was considered, as the neck swelling was thought to be separate and glandular, and possibly to contain secondary deposits. Surgical extirpation was advised, because the tumour, encroaching on the mouth space, was causing distressing symptoms.

Ether given by the colonic method[1] was used for anæsthesia, as it has given satisfaction in head operations in my practice for a score of years. Two hours before operation 0·25 grain of morphine in two millilitres of 50% magnesium sulphate solution was injected intramuscularly. At this time a ten-grain capsule of chloretone was inserted into the rectum, previously washed clean. After half an hour, and again in a further half-hour, two millilitres of the 50% magnesium sulphate solution (without morphine) were injected. One hour before operation six ounces of ether and two drachms of paraldehyde were mixed with three ounces of olive oil and given *per rectum*, some twenty minutes being spent in the introduction. The ears were plugged, the eyes were covered, and a hot, moist towel was placed over the nose and mouth.

With the help of Dr. Short, a curved incision was made in the neck. After the facial vessels had been tied off, a finger was inserted between the tumour and the buccinator muscle; the separation was continued more cranially and medially, and the tumour was wiped out of the palate. Neither the muscles of the cheek and palate, nor their mucous membranes were perforated. The tumour was removed by finger dissection from the base of the skull, and then finger dissection in a dorsal direction freed the tumour from the transverse processes of the atlas and axis and from the styloid process. The finger then removed the tumour from the muscles of the inner side of the mandible. The tumour was at this stage left attached to, or extended into, the carotid sheath in a confused line from the base of the skull caudally for about two inches. It was removed by scissor dissection and proved to be scltitary; it measured some five inches in length and about two inches in all diameters. The only artery requiring ligation was at the base of the skull near the *foramen spinosum*. No enlarged glands were found.

The Institute of Medical and Veterinary Science reported the tumour to be a mixed tumour of the palate, with endothelial sarcomatous changes. The patient is undergoing deep X-ray therapy.

Comment.

Ewing[2] states that undisturbed mixed tumours rarely invade lymph nodes, but after unsuccessful operation, while

recurrences are usually local, the cervical glands may be progressively involved. The fact that the therapeutic effects of radium are of much value in this group of tumours has a bearing on the wisdom of removal, which is often followed by recurrences of increasing malignancy.

References.

(1) J. T. Gwathmey: "Nelson's Loose-Leaf Surgery", page 525.
(2) J. Ewing: "Neoplastic Diseases", 1942.

Reviews.

INFECTIOUS DISEASES.

A VADE-MECUM has been described as a volume especially devised for projecting knowledge into minds not too inquiring or fastidious, so that a person who knows but little can appear to know a lot. Brevity is its virtue. But it behoves a compiler, in attaining that brevity, to be sure that what he puts down consists of the essence of the subject. That matter must be accurate and up to date, and he must not waste precious space. He must keep a sense of proportion, and if the range of subjects is wide, the information must be that which is most important to most of his readers. The difficulties did not deter the staff of the Cantacuzène Institute (Bucharest) from issuing a "revised and augmented second edition" of a "Handbook of Infectious Diseases"[1] originally published in 1939.

Certainly the book is concise. The pages are small and the lines short. Infections due to bacteria (including the Rickettsioses) are disposed of in seventy-seven pages. Each disease is dealt with under nearly identical headings: definitions, synonyms, ætiological agent, incubation, direct contagion, indirect contagion, portal of entry, localizations, symptoms, prognosis, channel of elimination, resistance of germ outside body, laboratory diagnosis, treatment, prophylaxis. With fine impartiality the answers to all of these are indicated; tularæmia is given almost as much space as tuberculosis.

In fairness it should be said that the statements set down under these headings are generally accurate so far as they go, though sometimes banal. In fairness also, the book was published in 1944, with the war within the country's borders. But the contents of this part I, and indeed of the whole book, are too uneven. We find pearls like "Tuberculosis—Incubation—Some weeks to some years". Just that! And again: "In diphtheria, as a rule the serum is injected subcutaneously under the skin of the abdomen, except in very grave cases." The page or so devoted to rheumatic fever might have been written thirty years ago. Cheek by jowl with quite admirable summaries of the serum treatment of acute staphylococcal infection and of tetanus we find details of treatment with certain antibacterial sera, as dead as the dodo, and vouched for by the ancient authorities of a past generation.

Streptococcal infection is treated unceremoniously. No mention is made of the work carried on, for some years just before the war, by Schwentker at Jassy. Yet Jassy is not far from Bucharest. Indeed the whole book gives the impression that information of advances in scientific knowledge, for the past decade at least, has reached the authors only occasionally and *en bloc*. Apparently they have been well informed of recent work on infective hepatitis and infectious mononucleosis, but the significance of streptococcal infection seems to have escaped them.

The chapter on virus diseases suffers from similar defects, but to a smaller extent. Within the limits it is adequate A quite original classification is attempted. The first class includes general febrile diseases, divided into groups with pustular, vesicular and macular eruptions, and septicæmic symptoms. In the second class are diseases with localization in the respiratory organs, the nervous system and other organs. On the whole the compilers have been made successful in this part of the work, and the description of such large groups as the encephalitides is reasonably clear. There is an interesting table of the Rickettsioses, grouped according to vectors.

Two chapters are devoted to infections by protozoa, metazoa and fungi; from malaria, hydatid and hookworm to scabies and favus.

Part II of the book is mainly technical. The principal biological tests are described, and the therapeutic and prophylactic uses of vaccines and serum occupy two chapters. Here and there the use of the blue pencil is strongly indicated, though there is little to cavil at as regards gross misstatement. The directions for vaccine treatment of whooping cough are at variance with those employed in Britain, America and Australia, and its use in prophylaxis is not even mentioned. Treatment of acute infections by injection of convalescents' serum in large doses has long been the vogue in Central Europe, and especially the Balkans, and is given prominence to a degree which is hardly fashionable at present. In some cases chemotherapy is mentioned almost as an after-thought, although towards the end of the book there is a short chapter on sulphonamides, with an addendum on penicillin. Here also is a quite good short description of the technique of transfusion and immuno-transfusion.

The faults of the book are that it is not always accurate, not always up to date, and, like all others of its kind, often out of perspective. Nevertheless the mistakes and omissions are not dangerous, and every reader will find something useful in at least one chapter. Many who are engaged in public health work will be interested throughout.

[1] "Handbook of Infectious Diseases, with Notes on Prophylaxis, Serum Treatment and Vaccination", by the Staff of the Cantacuzène Institute under the direction of Professor C. Ionescu-Mihăesti and Professor M. Ciucă; 1945. Geneva: League of Nations. Sydney: H. A. Goddard Proprietary Limited. 6" × 4¼", pp. 331. Price: 6s. 3d.

BACTERIOLOGY AND IMMUNITY.

IN laboratories throughout the British Empire, "Topley and Wilson", as it is usually known, has been used as a kind of bacteriological Bible ever since its first appearance in 1929. The second edition was published in 1936, and now after an interval of ten years we have the third.[1]

In the preface Professor Wilson tells of the special difficulties which beset its preparation, first the great loss of the senior author, then war conditions generally, and finally the limited access to international publications by which its bibliographies have suffered.

It is impossible in a review such as this to discuss all the new material which has been added to the text, but a few outstanding examples may be mentioned. The two-volume form has again been adopted, and there are several new chapters. The subject of chemotherapy, the mode of action of these drugs, and the theory of Woods and Fildes are discussed fully, also the antibiotic agents. New evidence of the existence of nuclear apparatus in bacteria is considered and admirably illustrated by some of Dr. Robinow's electron micrographs. The bacteriophage demands much more space than formerly; its antigenicity, its ecology and its application to the classification of the parasitized bacteria are recognized. The chapter on genus *Bacterium* has been divided into three. This gives more discussion to Shigella and Salmonella, and the section on the latter organism includes details of the ever-increasing types and their antigenic and biochemical constitution. In Parts III and IV, the systematic application of bacteriology to infectious diseases, a section on chemotherapy is included with each one, and already some of the text is outmoded. Sulphonamides are recommended as first choice for the treatment of gonococcal infections and penicillin second, and no mention is made of the definite dangers of under-treatment and the production of sulphonamide-resistant strains of the infecting organism by this means. Experiences in Australia and in other countries during the past two years have made it clear that penicillin is the most effective drug, and its use is attended with little risk of the development of drug fastness. The section on the bacteriology of the air is a new one, as yet small, but relevant work and principles of a subject which is receiving increasing attention on the part of public health and hospital authorities are well expounded. The classification of the viruses has been slightly rearranged and much modern work included. The bibliographical sections are very large, in order, as Professor Wilson observes, to balance some deficiencies in the text, though many continental and Japanese publications are not available.

The reader will be grateful that the familiar and well-loved tool of the laboratory worker appears in its same format and in the same blue covers, and that it has the same "feel". We are confident that in its pages relevant facts, references and discussions of the problems, great and small, which arise in the course of the day's work will be found with the same certainty as of yore.

[1] "Topley and Wilson's Principles of Bacteriology and Immunity", revised by G. S. Wilson, M.D., F.R.C.P., D.P.H., K.H.P., and A. A. Miles, M.A., F.R.C.P.; Third Edition; 1946. London: Edward Arnold and Company. In two volumes. 9¼" × 6¼". Volume I: pp. 1,028, with illustrations; Volume II: pp. 1,140, with illustrations. Price: 60s. net (two volumes).

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PENICILLIN IN THE TREATMENT OF SYPHILIS.

In December, 1944, reference was made in these columns to a preliminary report on an inquiry into the treatment of early syphilis by penicillin, instigated by the Committee on Chemotherapeutic and Other Agents of the National Research Council of the United States. Included in the discussions were the preliminary findings of a study carried out under the direction of the Subcommittee on Venereal Diseases of the National Research Council and reported in papers read at the annual meeting of the American Medical Association in June, 1944. It should be recalled that throughout that discussion stress was continually laid on the fact that all the work was preliminary and that it had to be treated as that and nothing more. J. E. Moore, who closed the discussion at the American Medical Association meeting, spoke on a "note of restrained optimism". He insisted that we were still in the process of learning how to use penicillin. A further report on the subject has now been issued; it is a joint statement by the Committee on Medical Research and the United States Public Health Service.[1] It is followed by another statement on the changing character of commercial penicillin from the same two bodies together with the Food and Drug Administration. These two reports are complementary to one another and they show beyond any doubt that the demand for caution in 1944 was more than justified.

No less than forty-one clinics and seven laboratories of experimental syphilis have cooperated to produce information for the first report. The clinics comprised four belonging to the army, one of the navy, thirteen of the United States Public Health Service Rapid Treatment Centres and twenty-three civilian clinics. The material on which the preliminary report was based has been used in this study, and in addition the effects of penicillin in the prevention of pre-natal syphilis and in the treatment of early infantile congenital syphilis, of certain forms of ocular syphilis and of neurosyphilis have been investigated. The study of latent syphilis and of cardio-vascular syphilis has been purposely avoided because they present long-term clinical problems of lesser importance. The information available up till August 1, 1945, was used. At this date twenty-six different treatment schedules had been instituted. The patients were treated according to these schedules. Satisfactory data are not available for all the schedules. Of the twelve which have satisfactory data, all but two consisted of the administration of penicillin alone (the total dosage and the duration of treatment were different in each instance), and the remaining two included the administration of arsenoxide in one instance and of bismuth in the other. The total number of patients treated by the cooperating clinics was 11,589, and these suffered from early syphilis; of this number, 10,482 (90·4%) were treated according to one or other of the schedules. The twelve schedules for which satisfactory data are available cover only 6,558 patients, or 56·6% of the total. Failures were defined as clinical relapse (muco-cutaneous or neuro-recurrence), serological relapse (blood or cerebro-spinal fluid) and reinfection. It is pointed out that the results of the use of penicillin in early syphilis must be interpreted in the light of the changing character of penicillin itself (of which more later), and we also need to remind ourselves that the dosage and duration of treatment (that is, the schedule adopted) must be continually borne in mind. With these provisos we may note that the cumulative percentage failure at the end of eleven months after treatment varied from 15% (when 2,400,000 units of the drug were given) to 62% (with 600,000 units). The cumulative percentage failure at the end of eleven months with a total dosage of 300,000 units was twice as great when penicillin was given alone as when it was given with 320 milligrammes of arsenoxide. The combination of drugs gave results the same as were achieved with penicillin alone at a total dosage of 1,200,000 units. The cumulative percentage failure at the end of four months with 1,200,000 units was twice as great when penicillin was given alone as it was when penicillin was given with 0·6 to 1·0 gramme of bismuth. When the disease was of long duration the failure rate was higher than when the duration was short. Thus when treatment was undertaken in the first week of the disease, the failure rate at the end of eleven weeks was half the rate noted when the disease had lasted two months or more before treatment was started. In about 85% of cases failure was thought to be due to relapse; reinfections probably accounted for 10% of failures. In regard to the failure of tests for syphilis to produce reactions with the serum—"evidence of seronegativity"—the results were interesting. The cumulative percentage of "seronegativity" by the end of the seventh month ranged from about 30 for patients receiving less than 60,000 units to about 50 for patients treated with 1,000,000 or more units on three hour seven and a half day schedules. Patients who were treated with 300,000 units of penicillin *plus* 320 milligrammes of arsenoxide had an accumulated seronegativity rate comparable to that obtained with the highest dosage of penicillin alone. Some anomalous findings were obtained with patients on three hour three and three-fourths day schedules. Data to allow an explanation of these findings

[1] *The Journal of the American Medical Association*, May 25, 1946.

to be sought were not adequate. When a schedule of 2,400,000 units was used the percentage of seronegativity at the end of seven months became less in those cases in which the duration of the disease increased before treatment was undertaken. When the disease had lasted two weeks before treatment was started, the percentage of seronegativity was 70; when the disease had lasted for more than eight weeks, the percentage was only 38.

It is necessary now to turn to the second report which in certain respects is the more important of the two. Since 1945 it has been known that there were four different types of penicillin, and these have been called, in the United States, penicillin G, X, F and K. These are identifiable chemical compounds, distinguished from one another by the side groups attached to a common nuclear structure. The proportions of these fractions may differ in the penicillin produced by different manufacturers and also from time to time in the penicillin produced by one manufacturer. These variations have occurred because of the use of various strains of *Penicillium notatum* or *Penicillium chrysogenum* and because of different techniques observed in the growth of the mould and in the purification of the final product. In addition to these changes in penicillin, there has been an increase in the purity of commercial penicillin. The penicillin originally used in the nation-wide study that has been mentioned had an approximate potency of 200 units per milligramme, and this gradually increased until at the time when the second report was written the potency was 900 to 1,400 units per milligramme. There has also been some evidence to suggest that some of the impurities removed might themselves have had treponemicidal properties. Experimental work has been carried out which shows differences in the effectiveness of the different kinds of penicillin in rabbit syphilis. Penicillin K is relatively ineffective in rabbit syphilis. The clinical results in the treatment of early syphilis have been less satisfactory since May, 1944, than they were before. Three possible explanations are suggested. The first is that there has been a sudden change in the biology of syphilitic infection. This is described as fantastically improbable. The second is that there has developed a strain of *Treponema pallidum* resistant to penicillin. This is regarded as improbable because no resistance in the same sense has been found to treatment by arsenic or bismuth. The third possible reason is that there has been a change in the character of commercial penicillin. We have already seen that this third possibility has actually occurred. Some references to recent work are quoted. Eagle and Musselman have shown that penicillin K is one-sixth to one-fifteenth as efficacious in pneumococcal and streptococcal infections in mice as are penicillins G, F and X. Holby has also found that penicillin K is only half as active as penicillin G against streptococcal infections in mice. Several groups of observers have also shown that penicillin K, unlike the other varieties, is largely destroyed in the body.

That intensive research is being undertaken to overcome the difficulties that have been mentioned will be readily understood. The matter is of the greatest importance. A discussion on the practical aspects will be found in the *Proceedings of the Royal Society of Medicine* for June, 1946. James Marshall insisted, and quite rightly, that attention must now be turned to the search for a method suitable for ambulant patients. Under the older methods of treatment a patient often received inadequate treatment and was chiefly a danger to himself. When rapid methods are used, however, and attempts to produce cure by a single dose are made, the patient is quite likely to become a danger to the community. Marshall observed that "experience has usually caused these rapid attempts to cure syphilis to become lengthened when the first flush of enthusiasm was spent". He added that "penicillin may be the exception, but it is too soon to say". Since penicillin, when it is used in the treatment of syphilis, must be used in the light of all available knowledge, it will be useful to mention some of the suggestions made in the second of the two reports. First of all, when sodium penicillin is used for the treatment of syphilis in man, injections should be given by the intramuscular route every two to four hours day and night round the clock for a minimum of seven and a half to eight days. The presence of penicillin K in commercial penicillin is compensated for by an increase in dosage and by a decrease in the time interval between injections. When relapses occur arsenic or bismuth is recommended in addition to penicillin. Secondly, it is stated that larger doses are required in the treatment of syphilis in infants. A third statement is that in no circumstances should penicillin in its present available form be given orally in the treatment of syphilis. Fourthly, the only satisfactory method for the absorption-delaying of penicillin is the administration of calcium penicillin in peanut oil and beeswax. Fifthly, it is suggested that 300 to 360 milligrammes of arsenoxide should be given intravenously in divided doses over a period of one to four weeks. Sixthly, bismuth may be given in addition to penicillin and arsenic. The suggestions named (neurosyphilis is omitted) are stated to offer a combination of medical desirability and expediency. Speakers at the Royal Society of Medicine discussion also insisted on the use of arsenic with penicillin. The conclusion of the whole matter is that, although a good deal of progress has been made in the penicillin treatment of syphilis, and although good results may be achieved if available data are carefully used, we still have a great deal to discover about the drug itself and about the way in which it may best be used.

Current Comment.

THE ANÆMIA OF INFECTION.

ANÆMIA occurring in and persisting after infective illness is one of the almost obvious of medical phenomena, but not very much is known about its exact mechanism. It has also been remarked that recovery from this type of anæmia is often slow and the reason for this has been thought in the past to be the lingering effects of the infection on blood formation. More recently the introduction of powerful anti-infective agents has revealed that this tardy restoration of a normal blood state does not always take place with much greater speed than in former days. G. E. Cartwright, M. A. Lauritsen, P. J. Jones, I. M. Merrill and M. M. Wintrobe have worked on this problem as a team and present a clear-cut picture of anæmia associated with infection, though they cannot add much as yet to our knowledge of its mechanism.[1]

The introduction of liver therapy for pernicious anæmia disclosed the great importance of coincident infection in this disease, and, though to a less extent, the same barrier

[1] *The Journal of Clinical Investigation*, January, 1946.

to recovery has been found in cases of simple microcytic anæmia. The hope that a better understanding of the processes underlying infective anæmias might lead to some specific method of treatment stimulated these authors in their inquiries. They point out that current experience as revealed in literature shows how common this type of anæmia really is. Infective endocarditis, rheumatic fever, pulmonary tuberculosis, sepsis and many other varieties of inflammatory disease all yield examples of coincident anæmia. The blood change is of the normocytic type in many cases, but may also be microcytic. Not many accounts were found of studies of the bone marrow. That increase in the leucoblastic activities might be found would be expected, but hyperplastic change has also been seen in the red-cell series. This latter change has not been associated with the appearance of undue numbers of immature red cells in the marrow. Though it would seem possible that additional blood destruction might occur with infections, there is little evidence of this, as estimations of the bilirubin content of the serum usually show no increase. Cartwright and his colleagues point out that there is also little critical information about blood volume in infective states, for the limits of variation are wide, though some writers suggest that a reduction in the blood volume may make the actual respiratory capacity of the blood lower than it appears from the figures seen in a blood count. Iron metabolism has been investigated by a number of workers who have shown that while the serum-iron content may be much reduced there may be evidence of retention of iron in the body possibly in the reticulo-endothelial system. Finally, all authors, whose labours have been thus briefly summarized, agree that treatment is difficult and disappointing. Even blood transfusion improves the rate of red cell regeneration rather than hæmoglobin synthesis.

Cartwright and his fellow workers have confirmed most of the above findings and have also concentrated on the alterations in iron metabolism. Not only does the value of the plasma iron drop greatly with infection, but they have found that the drop is surprisingly speedy. Even twenty-four hours after the onset of a severe infection a great fall may be demonstrated. From observations of a few patients it would appear that this fall precedes the onset of anæmia, if anæmia does occur at all, for no anæmia may appear in spite of the lowering of the iron content of the plasma. The same authors, working on dogs, found that artificially produced abscesses caused a rapid and sustained fall in the plasma iron, though a much less striking effect was produced by staphylococcal toxins. In human patients repeated observations showed that hypoferræmia remains as long as infection persists. Where anæmia was also observed the clinical condition and the blood counts of these patients were restored to normal long before the iron in the plasma rose to the usual height. Porphyrin studies were also made, but it was not found possible to correlate these observations with the other clinical data. The copper content of the serum was found to be raised. Various methods of treatment were tried, including the administration of iron intravenously, but no evidence of striking success was found until the causative infection was quelled. Whole blood transfusions, as might be expected, improved the well-being of the patients and alleviated the anæmia, but failed to make any real impression on the disturbances in the metabolism of iron or the porphyrins. The authors present some evidence that the underlying perversion of the body processes may be a failure to form "hæm" as the result of a lack of iron with which to build the protoporphyrin molecule, the iron being diverted to the tissues and thus made unavailable. This seems feasible.

The clinician, who so often uses the results of the researcher without thinking much of the long and hard road he has to travel, may think this is much ado about nothing, but he may yet be given an effective weapon to use against this rather refractory type of anæmia. Certainly more emphasis can now be laid on prevention, for with powerful drugs at hand to cut short some of the severe infective diseases and blood transfusion as a routine measure in daily practice, prompt measures should help to lessen those chemical changes in the body that make blood regeneration more difficult. The experience of the war years abundantly reinforces this lesson and civil practice should be the richer for it.

FOLIC ACID.

AN important announcement has been made by R. B. Angier and his fellow workers.[1] They have elucidated the chemical structure of the vitamin, folic acid. It has been shown to be a compound of glutamic acid, para-aminobenzoic acid and a pterin. The last-named compound is thought probably to account for the yellow colour of the pure vitamin, and the presence of three distinct growth-promoting substances in the compound to explain the conflicting reports on the nutritional significance of various concentrates of folic acid. It will be recalled that one of the components of folic acid (p-a-b) has for some time had a place in the vitamin *B* complex.

A NATIONAL COLLECTION OF CLOTHING FOR OVERSEAS RELIEF.

THE Australian Council for the United Nations Relief and Rehabilitation Administration, which is composed of forty-eight voluntary organizations, has decided to organize, in conjunction with UNRRA, a national clothing collection for 1946 for relief in China and other UNRRA countries. A successful drive was made in 1945 and even greater cooperation is sought this year. Last year the drive yielded more than 3,500,000 pounds' weight of very good clothing. At a conservative valuation of ten shillings a pound, this represents a total contribution of over £1,700,000 worth of clothing. This year it is intended that the greater part of the clothing collected shall be sent to China. The remainder will be sent to UNRRA countries which are still in great need. There is a world shortage of textiles, and it will be a long time before the distressed victims of war can hope to satisfy their needs from post-war production. This announcement is made in the hope that medical practitioners throughout the Commonwealth will do what they can to ensure success of the effort in their local centres. The organization in each State will be carried out by the UNRRA State Regional Officer, who is also responsible for the business administration at the following places: *New South Wales*: Sydney, 52, William Street, and Newcastle, Civic Hall; *Victoria*: Melbourne, 440, Collins Street; *Queensland*: Brisbane, 172, Edward Street; *South Australia*: Adelaide, c.o. the Principal, Parkin College, Kent Town; *Western Australia*: South Perth, 13, Riverview Street; *Tasmania*: Hobart, Mr. C. Murphy, Parliament House; *Australian Capital Territory*: Canberra, Miss Jones, Young Women's Christian Association. While the Directing Committee on the one hand lays stress on the importance of the greatest possible effort at collection, it points out that cash donations are not acceptable and that any organized purchase of new clothing for the drive is not to be encouraged. The need is urgent, and facts about the 1946 collection should be made known as widely as possible.

THE OTHER SEVENTY-FIVE PER CENT.

WHAT are the other 75% of members of the medical profession going to do about the Federal Medical War Relief Fund? This is the question which the Honorary Treasurer of the Federal Council asks in a letter published in this issue. The question is as urgent as it is pertinent. The need cannot and will not be completely met by payments from the State. Surely we cannot do less this time than was done after the earlier war. Money talks; here we should let it shout.

[1] *Science*, May 31, 1946.

Abstracts from Medical Literature.

PATHOLOGY.

Primary Carcinoma of the Fallopian Tubes.

ACCORDING to Karl A. Lofgren and Malcolm B. Dockerty (*Surgery, Gynecology and Obstetrics*, February, 1946), primary carcinoma of the Fallopian tube is a rare condition which accounts, in their experience, for only one out of every six hundred and twenty-five carcinomata of the female genital tract. For the most part it is a disease of the menopause and has not been reported among individuals in their prepubertal years. Symptoms are insidious in their onset with vaginal discharge, pelvic pain and abdominal tumour, these being cardinal symptoms which, however, rarely lead the physician to a correct pre-operative diagnosis. The evolution of the disease is more rapid than is indicated by its symptomatology, and the majority of patients so afflicted manifest lesions of doubtful resectability at the time of surgical exploration. The appearance of the lesion on gross inspection is sometimes deceiving, since it may mimic closely familiar inflammatory tubal conditions. Because of this every distended Fallopian tube should be opened before it is removed from the operating room. Treatment of these carcinomata, whenever found, regardless of any apparent state of encapsulation, should consist of total abdominal hysterectomy with bilateral salpingo-oophorectomy in addition to removal of any discernibly involved regional lymphatic nodes and peritoneal implants. Post-operatively X-ray therapy should be administered. In spite of these measures the number of five-year cures is disappointingly small. Pathologists who are unfamiliar with the condition will often have difficulty in establishing the primary nature of the lesion and should remember that the Fallopian tubes are frequently involved in metastasis from ovarian and uterine sources. Actual involvement of the tubal mucosa, strict confinement to the Fallopian tube, a marked disproportion between the size of the tubal and extratubal lesions, as well as a working knowledge of the histological pictures of primary uterine, tubal and ovarian carcinomata, will be helpful in deciding the primary source in doubtful cases. The epithelial hyperplasia associated with tuberculous salpingitis should be kept in mind, and the diagnosis of tubal tuberculosis and carcinoma should not be made except when malignant neoplasia is manifestly indicated.

The Origin, Frequency and Significance of Microscopic Calculi in the Kidney.

A STUDY was made by Leo Anderson and John R. McDonald (*Surgery, Gynecology and Obstetrics*, March, 1946) of microscopic calculi as found in the pyramids of 168 kidneys. These included surgically removed kidneys and grossly normal kidneys removed at necropsy. The diseased kidneys were removed for tuberculosis, hydronephrosis, pyelonephritis or stones. Six hundred and twenty-nine histological preparations were made. Originally only one hæmatoxylin and eosin section of each kidney was studied. Failure to find the microscopic calculi after study of the section from any particular kidney was followed by the cutting of further pyramids from the same kidney until the plaques were found or their absence was proved beyond a reasonable doubt. All of the kidneys were from patients more than nine years of age. In 50% of the kidneys the plaques were found in the first section studied. In the case of some kidneys, as many as ten histological preparations were made. The microscopic calculi were found in all of the kidneys. These microscopic calculi were studied by the authors as to morphology and possible histogenesis. It was demonstrated that they were formed by the coalescing of numerous "droplets" of calcareous material. These droplets seemed to be formed by the process of phagocytic ingestion of tiny "flecks" of black-staining material. Attention was directed to the postulate that the concentration of calcium is probably high in the tissue fluid about the renal tubules and to the fact that phagocytic cells occur in abundance about the renal tubules. From these data it was suggested that the calculi were formed by the process of phagocytic ingestion of the calcium which is reabsorbed by the renal tubules. It was shown that the tiny calculi occur throughout the renal parenchyma. Some were located just under the epithelium at the tip of the papilla, and one photomicrograph demonstrated the plaque ulcerating through this epithelium and becoming exposed to the urine of the calyces. It is now fairly well accepted that some renal calculi are formed by the process of the salts of the urine being deposited in layers on these plaques. Since a chemically pure calculus is very rare, the suggestion is made that perhaps many different types of stone may have this tiny calculus as a nidus. Since these microscopic calculi occur in practically all people, even though only one part in every 3,333 parts of the total pyramidal region of each kidney was cut and examined, the question was raised why more people do not form symptomatic stones. The interpretation was suggested that large stones may form only when some inciting factor comes to the kidney which causes the salts to be deposited on this eroded nidus plaque. Perhaps this inciting factor, substance or catalyst, the absence or presence of which is necessary to cause deposition to occur, comes from outside the kidney. From the foregoing data the proposal was made that perhaps renal calculus is a systemic or a dietary disease and not a disease of the kidney *per se*.

Acute Infectious Lymphocytosis.

ACCORDING to R. F. Birge and Lee F. Hill (*American Journal of Clinical Pathology*, November, 1945), a disease of children, named in 1941 by Smith acute infectious lymphocytosis, is characterized by pronounced absolute lymphocytosis of three to nine weeks' duration. Often it occurs without clinical signs or symptoms. Occasionally it is accompanied by slight fever, by symptoms of upper respiratory infection, by abdominal pain of short duration or by signs of meningeal irritation with slight pleocytosis. The disease is communicable and occurs at all seasons. Estimates of the incubation period vary from twelve to forty days or over. White and Negro children of both sexes are susceptible. The ætiology is not known. The prognosis is favourable. The disease seems to be a specific entity and differs from infectious mononucleosis in many respects. Lymphadenopathy and splenomegaly are not encountered; lymphocyte counts are increased; lymphocytes are normal in appearance; the result of the heterophile antibody test is negative. A typical case of acute infectious lymphocytosis illustrating the importance of not confusing the condition with leuchæmia is reported. Bone marrow obtained from a recently studied case showed slight increase in small lymphocytes, without other changes, confirming the observations of Smith.

Accidental Transplantation of Cancer in the Operating Room.

W. W. BRANDES, W. C. WHITE AND J. B. SUTTON (*Surgery, Gynecology and Obstetrics*, February, 1946) report a case in which, several months after a radical mastectomy for a highly malignant scirrhous carcinoma of the breast, cancer nodules were found in the skin of the donor site on the opposite thigh. The usual precautions had been taken after biopsy of the tumour and before the mastectomy, namely, changing of operators' and nurses' gloves, and redraping and re-preparation of the patient. However, after the mastectomy, when a defect in the skin over the operative site was to be covered, a skin graft was taken from the left anterior thigh without the contaminated gloves used at operation first being changed. Washing the gloves in sterile distilled water was apparently insufficient, and tumour cells were transferred from the operative to the raw donor area, where they survived and gave rise to tumour nodules. Previous work in which tumour cells have been demonstrated on knives used for biopsies is reviewed, and it is also shown that the fluid customarily used in the operating room to wash the gloves of the operators is another potential source of contamination of distant parts with malignant tissue.

Malignant Granulosa Cell Tumour with Pseudotubercles.

HERBERT J. SCHATTENBERG AND W. H. HARRIS (*The American Journal of Pathology*, May, 1946) report a case of granulosa cell tumour which satisfied some of the criteria for malignancy in neoplasms of this group. It also presented the previously unemphasized histological finding of pseudotubercles within its structure. It is believed that the best explanation for the presence of pseudotubercles is that they represent a stromal reaction to lipids derived from disintegrating tumour cells.

Massive Islet Cell Tumour of the Pancreas without Hypoglycæmia.

SEATON SAILER AND M. M. ZINNINGER (*Surgery, Gynecology and Obstetrics*, March, 1946) report on a large tumour of the pancreas which was removed from a forty-eight-year-old woman who was known to have had a palpable mass in the abdomen for two or three years, but whose symptoms were those of duodenal ulcer. Study of the tumour

showed it to be an islet cell tumour of the pancreas. Gross findings at operation and microscopic study indicated that it was malignant. At no time were there any symptoms of hypoglycæmia and several glucose tolerance tests gave normal results. Local recurrence and widespread metastases occurred, and the patient died three years and ten months after removal of the tumour, the immediate cause of death being mesenteric thrombosis.

An Analysis of the Klippel-Feil Syndrome.

FROM an example of the Klippel-Feil syndrome and from the cases reported in the literature, C. A. Erskine concludes (*Archives of Pathology*, March, 1946) that the esential features of the cervical deformity are synostosis of two or more cervical vertebræ and flattening and widening of the vertebral bodies. A numerical reduction of the vertebræ is an incidental rather than an essential part of the disorder, as is *spina bifida*. The latter depends largely on the degree of abnormality of the vertebral bodies. There is evidence that the anomaly has a genetic basis. A number of pathological conditions which have been found in association with the osseous deformity of the syndrome receive an explanation in the light of recent observations in the field of experimental embryology.

MORPHOLOGY.

Nerve Fibre Diameters in Nerve Grafts.

W. S. HAMMOND AND J. C. HINSEY (*Journal of Comparative Neurology*, August, 1945) find that in the choice of nerve grafts, the diameters of the nerve fibres should not be less than those of the host nerve. Regenerating myelinated fibres may be limited by the smallness of "Schwann tubes" into which they grow, and they will not exceed their normal size if large tubes are available. Vacant "Schwann tubes" and free sheath cells are not necessary as a pathway for regeneration of nerve fibres. Orientating effects of a nerve trunk facilitate regeneration. A regenerating nerve will recover to approximately 80% of normal distal to a suture after 365 days. The proximal segment of a regenerating nerve may show a loss of median fibre size after suturing. This loss bears a direct relation to the number of recurrent fibres shunted back and may be an index of the excellence of the suture. Individual normal fibres probably will not show any significant diminution in diameter.

Central Pain Paths.

E. GARDNER (*Archives of Neurology and Psychiatry*, June, 1945) studied the spinal cord and brain stem of a tabetic patient who died twenty-one days after a bilateral chordotomy. The chordotomies were performed at the sixth thoracic segment on the left side and between the fourth and the fifth thoracic segment on the right side. Subjective relief of pains in the legs and gastric crises were obtained, and there was loss of pain and temperature sense up to the sixth thoracic dermatomal area on the left side and to the seventh thoracic dermatome on the right side. Decussation of the tracts was thus observed to be completed within two segments, and the degeneration, as shown by the Marchi technique, was almost bilaterally symmetrical in ascent. The degeneration included lateral spinothalamic, spinotectal and dorsal and ventral spinocerebellar fibres. Relative lamination was confirmed by the increasingly superficial course of the fibres in the cervical part of the cord and the lower portion of the medulla and their position dorso-lateral to the inferior olivary nucleus. Incorporation of the lateral spinothalamic tract into the lateral lemniscus and the area at the base of the inferior colliculus, its entrance into the *nucleus ventralis posterolateralis* of the thalamus and the relative scarcity of degenerating fibres here were also established.

Argyll Robertson Pupil.

M. B. BENDER (*Archives of Neurology and Psychiatry*, June, 1945) states that after an oculomotor ophthalmoplegia the iris may be found to contract on convergence but not to light, thus simulating the Argyll Robertson pupil. Closer examination, however, discloses that this pupil reacts not only on convergence, but synkinetically with other movements, namely, downward, inward or upward movement of the affected globe. The synkinetic pupillary constriction may sometimes be associated with synkinetic retraction of the lid. The phenomena of synkinetic pupillary constriction and retraction of the eyelid can be reproduced experimentally by intracranial section of the oculomotor nerve in the monkey. These phenomena are partly explained by the theory of indiscriminate regeneration of oculomotor nerve fibres. It is concluded that the so-called or pseudo-Argyll Robertson pupil, sometimes noted after head injury with a complicating oculomotor ophthalmoplegia, has no ætiological relationship to the classic pupillary reaction originally described by Argyll Robertson.

Comparison of Action of Extension of Knee and Elbow Joints of Man.

H. HAXTON (*The Anatomical Record*, November, 1945) records that by the use of tensometers on the limbs of cadavera, it has been found that in the knee and elbow joints the leverage of the action of the extensor muscles on the joints becomes greater as the joints are extended from the flexed positions. The increase is greater in the knee than in the elbow, and it extends over a greater range of the movement of the joint. These findings are explained by reference to the anatomical features which determine the relation of the joint axes to the extensor tendons. The patella makes an important contribution to the leverage of the extensor apparatus of the knee joint. The advantages and disadvantages of the different mechanisms at the knee and elbow joints are discussed.

Development of the Human Lateral Geniculate Body.

E. R. A. COOPER (*Brain*, September, 1945) finds that the human lateral geniculate body (*pars dorsalis*) differentiates in the lateral thalamic nucleus, while the *pars ventralis* is subthalamic in origin. The *pars dorsalis* is recognizable in the embryo of 22 millimetres, and arises as the optic tract reaches this region of the thalamus. It is the first nuclear mass to be distinguished in the thalamic portion of the diencephalon. The *pars ventralis* is first seen at 35 millimetres. No lamination is evident until the sixth month of gestation. From the outset there are six U-shaped and V-shaped laminæ which present their closed convex aspects ventro-laterally and receive the fibres of the optic tract. From the dorso-medially directed concavities issue the optic radiations. The four larger outermost laminæ consist of small cells, while the two smaller innermost laminæ contain large cells. There is no such thing as "eversion" of the laminæ in the human or monkey lateral geniculate body as originally described and figured by Clark and his associates. The laminæ are curved to a lesser degree in the lemurs and to a greater degree in man and the monkeys, but all are curved in the same direction, the convexity always receiving the optic tract.

Innervation of the Pancreas.

C. A. RICHINS (*Journal of Comparative Neurology*, December, 1945) finds that all the nerves of the cat's pancreas pass through the cœliac plexus and reach the gland via the blood vessels which supply it. Just peripheral to the cœliac ganglia, myelinated and non-myelinated fibres are present in a ratio of approximately 1 to 19. The sympathetic preganglionic fibres terminate exclusively in the cœliac ganglia and aberrant ones which tend to spread along the pancreatic blood vessels. The parasympathetic preganglionic fibres terminate in the intrinsic pancreatic ganglia which lie in the interlobar and interlobular connective tissue septa. Sympathetic post-ganglionic fibres pass through the intrinsic pancreatic ganglia, but establish no synaptic connexions in them and are distributed solely to the pancreatic blood vessels around which they form perivascular plexuses. Parasympathetic post-ganglionic fibres course along the blood vessels in small bundles and pass between the pancreatic lobules and around the acini for ultimate distribution to the acinar and islet cells. They send off fine branches with abundant small knob-like enlargements. The appearance of the nervous tissue is similar on both acinar and islet cells, and single nerve fibres effect contact with cells of both types. The knobs undoubtedly are points of physiological contact. Terminal loops are seen on the surfaces of some acinar cells, but they are not abundant. Parasympathetic post-ganglionic fibres are observed along the pancreatic ducts, but they are not related to the epithelium. Where smooth muscle is present around the ducts they innervate it. The most obvious sensory end-organs in the cat's pancreas are the Pacinian corpuscles.

Regeneration of Mammalian Striped Muscle.

W. E. LE GROS CLARK (*Journal of Anatomy*, January, 1946) records observations made on regeneration in pieces of striated muscle grafted *in situ* and in crushed muscle, on the degree and extent of regeneration which is found to have occurred several months after ischæmic necrosis and on the histogenesis of muscle regeneration.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 20, 1946, at the Royal North Shore Hospital of Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary medical staff of the hospital. Parts of this report appeared in the issues of July 27 and August 3, 1946.

Renal Dwarfism.

DR. S. G. NELSON showed a male patient, aged sixteen years, who had been admitted to hospital on September 21, 1937, at the age of seven years, suffering from restlessness, irritability, cough and gastro-enteritis. On examination of the patient, diuresis was found to be present, and it was thought that he might be suffering from *diabetes insipidus*. The diuresis was controlled by posterior pituitary extract, half a grain being given by mouth twice a day. He was discharged from hospital on October 31, 1937.

The child attended the out-patient department regularly for years. He was always much smaller than the other children in his class; he lost much of his time at school, and thus was older than the other children. Periodical blood counts revealed anæmia, which was partly relieved by "Hepasol"; the erythrocytes were shown to be not unduly fragile.

He was admitted to hospital on March 5, 1946, suffering from anæmia and polyuria. It was thought that the anæmia might be of the aplastic type; the fragility of the erythrocytes was normal. The systolic blood pressure was 150 millimetres of mercury and the diastolic pressure 100. He was treated with an aluminium acetate mixture and an alkaline powder, and discharged from hospital on March 31, 1946. At the time of the meeting, his weight was six stone and his height was four feet ten inches. The systolic blood pressure was 110 millimetres of mercury and the diastolic pressure 60. The urine was alkaline and contained a cloud of albumin; the specific gravity was 1·004. Clinical improvement was being maintained, and a urological investigation was being carried out.

A number of investigations were carried out on this patient. On March 11, 1946, the blood urea level was 224 milligrammes *per centum*, and on March 16 the figure was 266; on March 16 the blood creatinine level was 3·5 milligrammes *per centum*. On March 14 a urea concentration test was carried out; a maximum excretion of 0·95 *per centum* was recorded after three hours. On March 19 the carbon dioxide combining power of the blood was 26 volumes *per centum*. No albuminuria was present on March 18; on that day the amount of lead excreted in the urine was estimated at 0·04 milligramme per litre, and no porphyrinuria was present. A glucose tolerance test was carried out on March 19; 25 grammes of glucose were given by mouth; half an hour later the blood sugar level was 200 milligrammes *per centum*; one hour after the ingestion of glucose the figure was 225 milligrammes *per centum*; one and a half hours after the ingestion of glucose the figure was 175 milligrammes *per centum*; after two hours the figure was 125 milligrammes *per centum*. On March 20 the blood cholesterol content was 160 milligrammes *per centum*.

The blood was investigated on a number of occasions. A blood count on September 21, 1937, gave the following results: the hæmoglobin value was 54%, the erythrocytes numbered 4,300,000 per cubic millimetre and the colour index was 0·63; the leucocytes numbered 6,200 per cubic millimetre, 49·5% being neutrophile cells, 45% lymphocytes, 4% monocytes, 2% eosinophile cells and 0·5% basophile cells. On February 27, 1946, a further blood count gave the following information: the hæmoglobin value was 46%, the erythrocytes numbered 2,280,000 per cubic millimetre and the colour index was 1·0; the leucocytes numbered 4,200 per cubic millimetre, 53·5% being neutrophile cells, 1·5% eosinophile cells, 1·5% basophile cells, 35% lymphocytes, 8·5% monocytes; platelets numbered 182,400 per cubic millimetre. Slight to moderate poikilocytosis and occasional polychromasia were present. The depression of the erythrocytes, leucocytes and platelets suggested some condition causing generalized aplasia of the bone marrow, and the question of exposure to some chemical such as benzol was raised. On March 14, 1946, the blood sedimentation rate was eighteen millimetres in one hour.

Chloroma.

DR. E. B. DURIE discussed a case of chloroma. This case will be reported in full in a later issue of the journal.

Basal-Cell Carcinoma of the Left Cheek, Involving Maxilla, Nose and Antrum.

DR. BASIL RILEY showed a male patient, aged fifty-nine years, who had suffered from a basal-cell carcinoma of the left cheek involving the maxilla, nose and antrum. It had been unsuccessfully treated by radium, and later the soft tissue and bone involved were excised and restoration was carried out by means of a rotation flap and an acromio-pectoral tubed pedicle graft, the latter supplying the lining of the antrum and the mucous membrane.

Squamous-Cell Carcinoma of Skin, Mucous Membrane and Cartilaginous Septum of the Nose.

Dr. Riley also showed a female patient, aged sixty-two years, who between the ages of fourteen and twenty-four years had in England been treated, apparently successfully, with X rays and tuberculin for suspected *lupus vulgaris*. In 1945 a similar condition reappeared on the nose, and was treated with carbon dioxide snow. On the patient's admission to hospital in March, 1946, her nose was almost entirely replaced by a large, foul-smelling, fungating mass. This, with portion of both cheeks, the vomer and the septum was excised; examination of all the excised tissue showed that the nose appeared to be affected by squamous-cell carcinoma. Dr. Riley said that reconstruction was being carried out by means of a lined forehead flap, in which had been embedded a framework of tantalum sheet, 0·005 inch in thickness.

Burns.

Dr. Riley also showed a male patient, aged twenty-two years, who had suffered the destruction by molten lead of two-thirds of his lower lip. Reconstruction was carried out by rotation of a right naso-labial graft, formed to supply a hairless lining, and an acromio-pectoral tubed pedical graft of the outer lip. At the time of the meeting (twelve months later), the stage of rotation of the naso-labial flap had been reached, and it was difficult to distinguish between skin and mucous membrane.

Radiation Necrosis of the Jaw.

Dr. Riley then showed a female child, aged thirteen years, who had been treated by deep X rays for a birth mark on the left side of her face. Dermatitis and ulceration of the left side of the cheek were present, together with fibrosis of the entire left masseter and buccinator muscles and impairment of development of the left facial nerve. These had resulted in ectropion of the left eye, paralysis of some of the facial muscles, gross displacement of the left angle of the mouth and extreme limitation of movement of the lower jaw. Efforts had been made to restore function by incising the masseter muscle from the buccal approach, thus enabling the mouth to be widely opened by means of a dental prosthesis and inlay graft. Much improvement was achieved. However, fibrosis had occurred, and it would be necessary to excise the entire masseter muscle. This procedure would create an extensive gap in the mucous membrane, subcutaneous tissue and skin covering, which would be replaced by means of an abdominal tubed pedical graft carried on the wrist.

The next patient shown by Dr. Riley was a male, aged sixteen years, who had had a birth mark, approximately two inches by three inches in area, involving the left side of the nose and the upper side of the left cheek. This had been treated by radium. Dense mottling of the skin had resulted, with areas of ulceration. The whole area was excised, and the defect was partly covered by a large Estlander rotation flap; the nasal portion was covered with a Wolfe graft. Dr. Riley said that the case illustrated the many advantages of the local flap method (when available) over any of the other methods used in the repair of facial defects.

Acanthotic Nævus.

Dr. Riley's next patient was a male, aged nineteen years, who had had on the left frontal region and involving the left eyebrow and left upper eyelid a large, raised, rugose, pigmented, acanthotic nævus mole measuring five inches by three inches and covered with coarse hair. This had been excised and replaced by a dermatome graft 0·6 inch thick from the abdominal wall. The eyebrow and portion of the upper eyelid had been replaced by a post-auricular full-thickness graft which included a narrow hair-bearing strip.

Trauma to the Nose.

Dr. Riley's next patient was a female, aged nine years; in her infancy the end of her nose and the tip of her fingers

had been bitten off by a parrot. Reconstruction had been carried out by means of a tubed pedicle graft raised on the inner aspect of the left arm, and transferred to the nose and maintained by means of fixation of the arm to the head by a plaster cap and gauntlet.

Bilateral Compound Fracture of the Tibia and Fibula.

DR. KATHLEEN CUNINGHAM showed a male patient, aged twenty-three years, who had been admitted to the casualty department at 7.30 o'clock p.m. on March 23, 1946. He had been driving a motor truck when the vehicle left the road and went over a cliff. He had sustained a compound comminuted fracture of the tibia and fibula in both legs and extensive and gross laceration of all soft tissues, especially in the left leg. Bone was exposed in both legs. He was in a state of profound shock, sweating and semi-conscious; his pulse rate was 104 per minute, and his systolic blood pressure was 70 millimetres of mercury; the diastolic pressure could not be estimated.

He received immediately by transfusion one litre of plasma, followed by one litre of blood. Three hours later his condition had improved sufficiently for a surgical reduction to be performed. Under anæsthesia both fractures were reduced simultaneously by Dr. Cuningham and Dr. A. R. Hamilton. The patient's condition was so poor that full *débridement* was impossible. In the left leg, the whole of the skin completely encircling the calf muscles was torn down in a flap about eight inches long, which appeared devitalized. The calf muscles were torn and the anterior tibial group was grossly lacerated. The left tibia was fractured in three places, and about one inch of this bone had to be removed. The fractured ends were bound together with kangaroo tendon. The left foot was cold and blue, and no pulsation could be felt in the *dorsalis pedis* artery. It was extremely doubtful whether the circulation could be restored in this leg and foot; but as the laceration of the soft tissues extended to behind the knee, and most of the skin on the anterior aspect was missing, any amputation would have necessitated a guillotine amputation through the lower end of the femur if the patient's condition improved. It was decided to attempt repair with plaster of Paris. This was done as rapidly as possible. The whole wound was cleansed with hydrogen peroxide and saline solution and dusted with penicillin and sulphanilamide powder. When the flaps were sutured a diamond-shaped gap was left on the anterior aspect, roughly six inches long and four inches wide with bare tibia in its base.

In the right leg, the soft-tissue damage was not so extensive as in the left leg, but it extended round the circumference of the limb. Suture was preceded by the same toilet as for the left leg, and in this case enough skin was found to cover the whole of the injured area. Both legs were encased in plaster of Paris with about 30° of flexion of the knee joints. The patient received a further litre of blood by transfusion, and sulphonamide and penicillin therapy was given.

On May 8 the plaster casts were removed. The right leg required further suture. The soft-tissue gap in the left leg was filled in with healthy granulation tissue for about half of its extent. The limbs were replaced in plaster casts. On May 28 the plaster casts were again removed. The right leg was completely healed, with firm callus formation; there was slight backward displacement of the lower fragments, but the callus was too firm to allow any alteration in position to be made. In the left leg, healthy granulation tissue covered all but the centre part of the damaged soft tissue; there was about one inch of exposed bone with healthy granulations in places. Some epithelialization had taken place. The circulation in the feet was good. There was some weakness of dorsiflexion of right toes in the right foot, but the patient's general condition was satisfactory. On June 20 the plaster casts were removed.

Mesenteric Cyst.

Dr. Cuningham also showed a female patient, aged twenty-six years, who had noticed a lump in the left side of her abdomen in January, 1942; the lump was not painful. The patient was married and had one child. She had suffered from erosion of the *cervix uteri*, which was treated by cauterization in 1945. Blood had been passed in the stools from 1942 to 1944. On September 20, 1944, a sigmoidoscopic examination was made; a simple ulcer was seen fifteen centimetres from the anus; no hæmorrhage occurred subsequently.

In March, 1945, the patient attended the out-patient department complaining of constant pain in the left hypochondrium and of a palpable mass in that area. She suffered from slight nausea. Examination revealed no mass when the patient was recumbent, but when she was standing a rounded mass could be felt under the costal margin; this mass slipped away under the fingers. A plain X-ray examination of the renal tract revealed no abnormality. Excretion pyelography showed that the right kidney was low in position; the pelvis was slightly dilated, but function was good. The left kidney was normal in position and its function was good. The leucocytes numbered 5,600 per cubic millimetre, 54% being neutrophile cells and 2% eosinophile cells; no abnormality was detected in the leucocytes. In view of the fact that the patient had lived in the country until the age of twelve years and owned a cattle dog, Casoni and hydatid complement-fixation tests were carried out; both produced negative results. At the time of the meeting the mass was still palpable. The provisional diagnosis was cyst of the mesentery or the jejunum.

(To be continued.)

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioner has been released from full-time duty with His Majesty's Forces and has resumed civil practice from the date mentioned:

Dr. D. L. Graham, Adelong, New South Wales (June 28, 1946).

A MEETING of the Section of Neurology, Psychiatry and Neurosurgery of the New South Wales Branch of the British Medical Association will be held at 2.30 p.m. on Thursday, October 17, 1946, at the Mental Hospital, Parramatta. The meeting will take the form of a clinical afternoon, and will be arranged by officers of the Division of Mental Hygiene, Department of Public Health of New South Wales.

Correspondence.

THE EPIDEMIOLOGY OF INFECTIOUS DISEASES.

SIR: One of the great problems in the epidemiology of infectious diseases is the mechanism by which infection survives through long interepidemic periods.

Based on the instability of the molecular structure of viruses, the solution of this problem is here suggested.

It is noteworthy that human epidemics usually occur during a particular season of the year, and that epidemic foci often arise simultaneously and apparently independently, suggesting some activation of previously acquired infection rather than direct case-to-case transmission.

In his study of swine influenza, Shope (1943) has demonstrated that swine influenza virus, while in the lung-worm, exists in a masked form and is totally non-infective until awakened to activity by some provoking stimulus. Shope, moreover, considers that the onset of a swine epizootic is determined not by the acquisition of the causative virus, but by meteorological or physical conditions which favour its activation.

A similar conclusion was also arrived at many years ago by Tomb and Maitra (1926-1927) regarding the relationship of "non-agglutinating" (non-epidemic) intestinal vibrios to epidemic cholera. In an article entitled "A New Conception of the Epidemiology and Endemiology of Cholera" (1927) they wrote as follows:

> We have therefore been driven to the unavoidable conclusion that the non-agglutinating (non-epidemic) vibrio takes on the agglutinating (epidemic) characteristic under certain biochemical-physical conditions [often seasonal] in the human intestine, the nature of which are at present unknown, and in this mutation or epidemic form is the cause of epidemic cholera. Non-agglutinating (non-epidemic) intestinal vibrios therefore in our opinion constitute the reservoir of cholera both epidemic and endemic.

The mechanism by which infectivity is increased or diminished in a virus is thus described by Fenton (1945):

> Viruses x x x are giant molecules containing hundreds of thousands of atoms that are linked in complex groups and series. For generations these molecules may remain stable. Then they abruptly change. Thus the

virus which normally causes tobacco mosaic may add a few thousand atoms and become a variety that produces the much more virulent ailment known as acuba. Other modifications may cause a disease that kills the affected plant [outright] or make the virus so weak that its effects can barely be detected. Apparently a single virus strain may appear in any or all of these forms, shifting from one to another as atoms are gained or lost.

An analogous mechanism in bacteria also may not unreasonably be inferred.

The variations in virulence which commonly occur in epidemic diseases are well illustrated by the history of scarlet fever.

According to Singer (1928), scarlet fever first became clearly recognizable as a mild disease without prominent symptoms about 1650. Good observers in the half-century which followed considered it a new disease. In England it continued to be of little importance till about 1748, when it began to be associated with grave throat symptoms. This phase continued for about ten years.

The virulence then dropped and the disease continued of slight importance till 1785. It then grew virulent again and remained so until about 1808. The malignancy then fell and remained low for about thirty years. It rose again about 1837, and from then till 1894 it was one of the great killing diseases especially of children.

Since then the mortality from it has steadily decreased.

Yours, etc.,

Sydney,
New South Wales,
August 14, 1946.

J. WALKER TOMB.

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THE "DRUNKEN" PERSON AND THE MEDICAL WITNESS, IN THE EYES OF THE LAW.

SIR: The medical man is most often called in as a witness by the police in cases of "drunkenness" and always so when a person has been arrested for being in charge of a vehicle while "under the influence", particularly if it be a motor-car.

The medical man comes to be the chief witness and a great responsibility is apparently placed on him and the evidence that he gives. In the witness box he is always subjected to a medico-legal battle or controversy which usually becomes quite a "heated" one.

From my personal experience in such cases the medical witness can cause or effect all the "heat" to be on the side of the defending counsel when, after his examination of an accused person, he has found him to be under the influence of alcohol or intoxicating liquor.

In my opinion the medical witness should refrain absolutely from allowing himself to be drawn into a controversy about "drunkenness" and its meaning *et cetera*, and he should therefore not use the word in his evidence.

The best definition of the word "drunkenness" was made by a very representative committee appointed by the British Medical Association in 1925 "to consider and report on the tests for 'drunkenness' with recommendations as to their modifications or improvements". And the definition of the word "drunkenness" was the outstanding finding of this committee, namely: "That the person concerned was so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation in which he was engaged at the material time."

The simple procedure then is for the medical witness (*a*) to establish the fact that alcohol has been consumed, and (*b*) to determine whether or not the amount of alcohol taken has disturbed the normal behaviour of the accused. And, covering (*a*) as having been established and (*b*) as having determined that the normal behaviour of the accused has been disturbed, by giving all his reasons and observations therefor, he can then complete his evidence by stating that the person concerned was so much under the influence of alcohol as to have lost control of his faculties to such an extent as to render him unable to execute safely the occupation in which he was engaged at the material time.

By following this procedure the medical witness has, in my opinion, given to judge or magistrate all the evidence that the latter requires from him, for, after all, the judge bases his judgement on all the evidence that finally he will have before him.

Defending counsel will always lay great stress on the degree of "drunkenness" and will cross-examine the medical witness on this point. But, by the medical witness avoiding the use of the words "drunk" and "drunkenness" he can materially shorten such a controversy.

An estimation of the percentage of alcohol in a blood-alcohol or urine-alcohol test, we all know, cannot be accurately assessed because of all the loopholes and pitfalls in such a procedure. Actually, in my opinion, the police are much more competent to judge the degree of "drunkenness" than is a medical witness. Also, in my opinion, the degree of "drunkenness" should be no concern of the medical witness, or certainly, only a minor concern, once he has given proof that the accused was so much under the influence of alcohol that he could not execute safely the occupation in which he was engaged at the material time, whether that occupation had been pushing a wheelbarrow, splitting wood or climbing upstairs *et cetera*.

The medical witness may be asked by defending counsel a question something like this: "Do you consider from your findings that the accused was unfit to drive a car?" The medical witness need only reply: "Yes."

And, furthermore, in my opinion, whenever and wherever possible two independent medical witnesses should be called in at the same time to examine a person who has been charged by the police with "drunkenness".

Yours, etc.,

Belgrave,
Victoria,
August 19, 1946.

A. L. J. PETERS.

THE FEDERAL MEDICAL WAR RELIEF FUND.

SIR: On February 11, 1946, the President of the Federal Council sent out his circular letter to all members of the British Medical Association in Australia appealing for contributions to the above fund. This was followed by your excellent leader in THE MEDICAL JOURNAL OF AUSTRALIA of February 16, 1946.

To date the sum of £15,000 has been subscribed. The Federal Council asked for £50,000, and this should not be too much for an association whose membership on January 1, 1946, totalled 5,861.

It may be early yet to comment on the response to the President's appeal, but one cheering fact stands out, and that is the generous way in which the service medical officers have contributed. At first the Federal Council felt in grave doubt about sending the circular letter to them, but it was felt that there may be some who would wish to give to a fund whose main object was to help the widows and children of their deceased comrades.

To date about 25% of the profession have contributed, and the amount collected is 30% of the objective. The old Medical Officers' Relief Fund (Federal) did a magnificent job, and is still functioning after twenty-eight years. It lent £23,500 to forty-seven medical officers, gave away in medical benefits nearly £10,000, and is still helping seven dependants of deceased medical officers, and is being repaid loans advanced to five ex-service men of the 1914-1918 war.

Up to the present time eighty-five medical officers have died while on full-time duty in the services, and the majority of these were young men who had had little chance of making provision for their dependants.

The earning power of invested trustee funds is only half that of the old fund, and consequently the amount asked for is not too much if an attempt is to be made to help to the same extent as the old fund.

Might I urge the 75% of our members who have not yet contributed to seriously consider the claims of this fund and to give promptly.

Yours, etc.,

W. F. SIMMONS,
Honorary Treasurer, Federal Council of the British Medical Association in Australia.

August 29, 1946.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Knight, Lindsay Wescombe, M.B., B.S., 1945 (Univ. Melbourne), Royal Hobart Hospital.
Coats, Howard Presswood, M.B., B.S., 1945 (Univ. Melbourne), Royal Hobart Hospital.
Rumbold, Sydney James, M.B., B.S., 1940 (Univ. Melbourne), Launceston.
Pyper, John Clark, M.B., Ch.B., 1912 (Univ. Glasgow), Ulverstone.
Etheridge, William Napier, M.B., B.S., 1945 (Univ. Melbourne), Zeehan.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Ackland-Horman, William Dinwoodie, M.B., B.S., 1941 (Univ. Adelaide), University Club, Sydney.
Frank, Nathaniel, M.B., Ch.B., 1933 (Univ. Glasgow), B.Hy., D.P.H., 1943 (Univ. Durham), c.o. Quarantine Service, Commonwealth Health Department, Sydney.
Hammond, Henry Grant, M.B., B.S., 1944 (Univ. Adelaide), District Hospital, Broken Hill.
Harrold, Arthur George, M.B., B.Ch., 1943 (Univ. Cambridge), c.o. P.O. Box 59, Moss Vale.
Kewish, Orme Kay, M.B., B.S., 1940 (Univ. Melbourne), Balranald.
Rothfield, Neville Jacob Harris, M.B., B.S., 1946 (Univ. Melbourne), Lithgow District Hospital, Lithgow.
Valese, Peter, M.B., B.S., 1942 (Univ. Sydney), 4, Brown's Avenue, Marrickville.

The undermentioned additional qualification has been registered:

Dalton, William Redmond, 171, Avoca Street, Randwick (M.B., 1941, Univ. Sydney), B.S., 1946 (Univ. Sydney).

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1940*, of Queensland, as duly qualified medical practitioners:

Uhd, Karl Neilson, M.B., B.S., 1936, M.D., 1940 (Univ. Melbourne), General Hospital, Brisbane.
Merton, Thomas Allenby, M.B., 1945 (Univ. Sydney), District Hospital, Cooktown.
Rimmer, William David, M.B., B.S., 1945 (Univ. Sydney), Walters Street, Lowood.
Tonge, John Iredale, M.B., B.S., 1939 (Univ. Sydney), Department of Health and Home Affairs, Brisbane.
Guthaner, Ernst, M.B., B.S., 1942 (Univ. Adelaide), the Canberra Hotel, Ann Street, Brisbane.
O'Neill, Vennard Francis Joseph, M.B., B.S., 1944 (Univ. Sydney), 23, Walker Street, Townsville.
Taylor, Benjamin John, M.B., B.S., 1945 (Univ. Sydney), Mount Isa Hospital.
Matchett, Victor Lewin, M.B., B.S., 1945 (Univ. Sydney), General Hospital, Bundaberg.
Mackerras, Ian Murray, M.B., Ch.M., 1924 (Univ. Sydney), Nelson Street, Corinda, Brisbane.

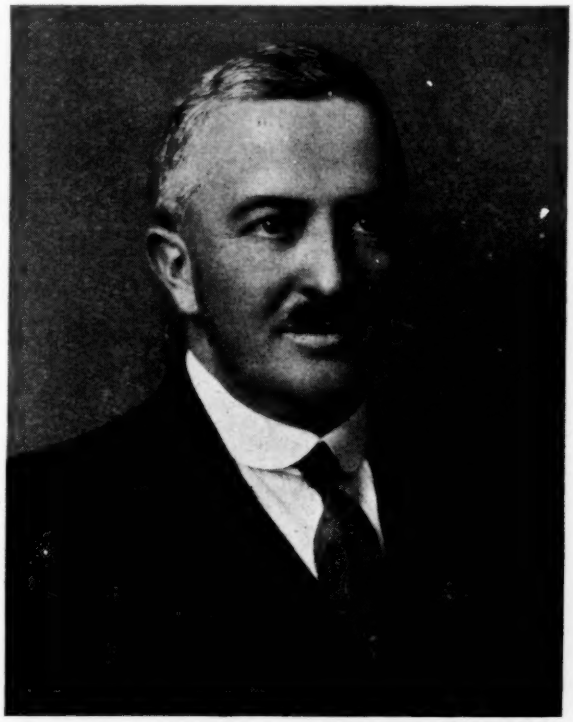
Obituary.

ROBERT MARSHALL ALLAN.

WE are indebted to Dr. E. R. White for the following account of the career of the late Dr. Robert Marshall Allan.

Robert Marshall Allan, Professor of Obstetrics and Gynæcology in the University of Melbourne, died on July 29, 1946, aged sixty years. He was a great leader and teacher in his speciality, not only in Australia, but abroad. He had a serious illness late in 1944, and next year he was compelled to rest from active work for a considerable time. Feeling somewhat restored, he determined this year to resume his full activities again, with the knowledge that the end might come at any moment. He died, as he would have wished, in harness.

Marshall Allan was born in Brisbane and gained his early education at Brisbane Grammar School and Scots College, Sydney. He then studied medicine and graduated at Edinburgh University and later passed the examination as a Fellow of the Royal College of Surgeons, Edinburgh. He now devoted himself to the speciality, which really became his life's work, for he went to Dublin and studied at the Rotunda Hospital, where he became assistant master for some years. Then came the 1914-1918 war, when Marshall Allan, always a man of high ideals and action, promptly enlisted and joined the Royal Army Medical Corps. He was sent over to France in the early days as a medical officer in



the field and rendered splendid service in those dark and strenuous months. Later he was transferred to a field ambulance in Mesopotamia and took part in the campaign for the relief of Kut. Here again he did splendid work and was awarded the Military Cross. At the end of the war he returned to Brisbane and commenced practice in his speciality.

Soon afterwards, in 1925, he was appointed from amongst a large number of candidates to the position of Director of Obstetrical Research in the University of Melbourne. Under the terms of his appointment, Allan traversed the whole State of Victoria and consulted with every single medical practitioner and reviewed with each one the obstetrical work, an investigation that required great tact and knowledge, qualifications that he was fortunately well endowed with. After two strenuous years, he published his report on maternal and neonatal mortality and morbidity in the State of Victoria. (See THE MEDICAL JOURNAL OF AUSTRALIA, January 1, 1927, page 1.) His keen observations and practical recommendations created a most favourable impression on everyone, and there is no doubt that the implementation of his advice has been primarily responsible for the great improvement during the past twenty years in the practice of obstetrics in this State, as exemplified by the marked lowering of the figures relating to maternal and neonatal mortality and morbidity.

In 1928, Marshall Allan became the first occupant of the newly endowed Chair of Obstetrics and Gynæcology of the University of Melbourne. The new professor, with his customary zeal and enthusiasm, began what has proved to be the most important work of his career. He became a member of the honorary staff of the Women's Hospital, Melbourne, where the committee and honorary staff afforded him every facility and assistance in the teaching of students, nurses and post-graduates, for it was at this hospital that his clinical work was performed.

As he was interested in research and modern development in the art of obstetrics, both at home and abroad, he managed to make two further trips to observe further progress made in Great Britain, on the Continent and in the United States of America. He was made an Honorary Fellow of the American College of Surgeons, and later he became a Foundation Fellow of the Royal College of Obstetricians and Gynæcologists of Great Britain. In recognition of his great work and leadership in obstetrics, four months ago he became vice-president elect, an honour for the first time conferred on a resident outside Great Britain.

He was also a Fellow of the Royal Australasian College of Surgeons. He was particularly interested in British Medical Association affairs and filled with dignity the office of President of the Victorian Branch of the British Medical Association. He held office in the Queensland Branch before he came to Victoria. He also was a former President of the Gynæcological and Obstetrical Section. In 1943 he became Dean of the Faculty of Medicine in the University of Melbourne.

Soon after his arrival in Melbourne, he became actively interested in the Victorian Bush Nursing Association, both as a council member and later as honorary secretary.

During World War II he gave up any leisure time that he might have had by again assisting his country in part-time services as a medical officer in the Australian Military Forces.

The writer became acquainted first with Marshall Allan at a British Medical Association Congress in Melbourne soon after the war. When he became established in Melbourne soon after, there began a close association in work together at the Women's Hospital, which developed into a close and lasting friendship.

He was a man of fresh ideas, good opinions, and, most important of all, he was a splendid and impressive teacher who at all times would discuss a subject fully and quietly but firmly with the student, nurse or graduate in an endeavour to bring out the best in his auditors and to give them a confidence in their own ability to do good and useful work.

Though he was held in high regard and esteem by many friends, yet the circle of his really intimate ones was small; to those few was revealed a warmth of affection that will ever be held in tender memory.

In spite of his high qualifications and world-wide reputation, Marshall Allan was a very modest man; he was sincerely religious and an elder of his church.

To his widow and son and daughter do we sincerely offer our deepest and heartfelt sympathy.

DR. COLIN MACDONALD writes: Marshall Allan was a handsome man of well-proportioned build with silver hair and twinkling hazel eyes which would attract attention in any company. His background in the main was composed of a Colonial Scottish Presbyterianism, a full undergraduate life in the heyday of the Edinburgh Medical School, and a long and varied service in the First World War, in which he was initially attached to a battalion of the famous King's Royal Rifles, oldest of the rifle regiments. He dearly loved the British people and their institutions, and was never happier than when cementing professional ties with the homeland or New Zealand; thus the furtherance in Australia of the Royal College of Obstetricians and Gynæcologists was very close to his heart.

The professor held unequivocal views on those controversial subjects of politics, religion and international affairs, sometimes forcibly expressing them in unexpected places; but this apparent rigidity of opinion was really only a very translucent facade for his true qualities of sincerity, constancy, helpfulness and kindness, which many a student—post-graduate as well as undergraduate—had occasion to confirm joyfully when examination results were posted. In his desk at the Women's was found the counsel:

"Let me not live . . .

After my flame lacks oil to be the snuff of younger spirits."

An omnivorous reader, with a splendid memory, he must surely have been one of the most knowledgeable men in this country, though his wide acquirements were veiled by modesty and often shyness. Not an eloquent speaker, his exposition of a subject invariably contained all the relevant facts, presented in a proper perspective; pretension and snobbery—intellectual or social—in whatever guise, he was quick to denounce at any time or place. He was steeped in the fine tradition of amateur Rugby football, in which at school and university he had played a first-class game; one can remember how, during the last month of his life, being subjected at luncheon to gentle badinage when it was reported that an Australian Rugby player had an ear bitten off, with what gleeful zest—sick man though he was—he countered that the malfeasance had occurred under the League, and not his beloved Union, code of rules. Cricket he was quite content to leave to Neville Cardus and other gentle essayists, nor did he ever become enmeshed in the architectural nuances of symphonic music.

Marshall Allan will always be fondly remembered by those privileged to be counted among his friends, for much of his six decades was spent in those oft-forgotten acts of kindness and goodwill which so generously temper life's asperities.

But "Real worth requires no interpreter; the everyday deeds form its blazonry".

DR. D. GIFFORD CROLL writes on behalf of the Queensland Branch Council: R. Marshall Allan was honorary secretary of the British Medical Association (Queensland Branch) from 1920 to 1925. Those were the days in which we knew him so well, and few of us pictured our alert young secretary as the staid professor of obstetrics of later years.

He had somehow acquired a very large Airedale dog, and he used to complain that there was not sufficient room in his car for himself and the dog as well.

The Queensland Branch never had a better or more progressive secretary, and he helped to steer it through those developing years which ended in its incorporation as a registered company before he left. As honorary secretary of the Branch he was also honorary coadjutor secretary of the Australasian Medical Congress, Eleventh Session, held at Brisbane in 1920, and those who remember the success of that congress will appreciate the quality of his organizing ability.

He left us in 1925 to take the post of director of obstetrical research in the University of Melbourne, after a farewell dinner and presentation by members of the Branch. He has visited us since on many occasions, and we hope and believe that he regarded the period of 1920 to 1925 as some of the best years of his life. Valuable to us they undoubtedly were. We offer our deepest and most sincere sympathy to his widow and family.

DR. S. F. MCDONALD writes: Although Marshall Allan and I had been boys together at the Brisbane Grammar School, I had seen nothing of him until my return from overseas in 1920. From his family one had heard of his academic success in Edinburgh—of his appointment as assistant master at the Rotunda from 1911 to 1914; of his war service I had heard a little, but when I met him hunting for rooms on Wickham Terrace it was the first renewal of our friendship.

For several months thereafter we lived a "Box and Cox" existence in a small cottage on the site of what is now Wickham House—for professional rooms were as scarce then as today. Later we moved together to a new building and continued to see a great deal of one another during the following five years.

He had come back to a somewhat unfortunate situation (I speak as an onlooker), a city where the practice of obstetrics—except in a few rare exceptions—was considered entirely the province of the general practitioner, who might or might not be well equipped for such routine work. Many such men had done no hospital work since their student days, their ante-natal care was very perfunctory, and too often they took little notice of the fact that parturition is a period of agony and discomfort. Not infrequently the attitude of the doctor (with again certain notable exceptions) was that pain was an essential part of labour and that all they could do was to put a nurse, trained or untrained, in charge, arriving themselves just in time to be in at the birth, perhaps using a little anæsthetic to tide over the worst. "Gloving and scrubbing up" such as I had been taught to do even "on the district" (in other words, the north-west London slums) at Queen Charlotte's in 1914 was still by no means general, especially among the untrained or semi-trained nurses who conducted most of the local obstetric practice. (It was in the practice of one such that I saw my first case of *tetanus neonatorum*.)

As a contrast there were a number of what might be called obstetrical busy-bodies, who actively interfered with labour by every means from quinine to Cæsarean section. Their work afforded much food for conversation at a certain type of afternoon tea party.

To all such people Allan appeared as at least a threat. The idea that a man should be prepared to disregard the rest of his practice to spend a long time by a mere midwifery case was preposterous; "what did it matter if the perineum were a bit torn—it could always be stitched—on the other hand, what harm was there in a Cæsarean section done under good conditions—better for the mother and much better for the baby, if carried out at the slightest hint of danger?" Such was the view I actually heard expressed by one man.

Allan was by neither nature nor upbringing prepared to compound with his conscience over such practices. He thought they were wrong, and he said so in forcible idiom of Australian and Scots—and as such saying do, they came to the ears of those he criticized. The inevitable reaction took place; "who was this fellow", said they, "who had the impudence to come here and criticize his elders and betters?" So instead of his receiving the recognition that his experience and qualifications merited, he found himself rather cold-shouldered and pushed to one side. So his first two years in Brisbane, despite the support of such friends as the late Dr. R. A. Meek, were years of disappointment and irritation. Moreover, Allan had always the mind of the teacher and prophet and he felt the lack of students intensely. Certainly he had the gratification of knowing that among the more intelligent of the nurses who passed through his hands, his teaching was received with gratitude and enthusiasm.

But gradually matters improved—his patients appreciated the attention of a man who went to unlimited trouble on their behalf, and they brought others.

Meantime, too, he had become secretary to the Branch, where his vigour and ability made him more friends among his colleagues, so that his prospects were steadily improving.

So matters stood when in 1923 he had his chance of carrying out the survey of obstetric conditions throughout Victoria, a survey promoted by the Wilson Trustees.

He hesitated very greatly—it meant giving up the fairly assured position he had gained, but on the other hand, he felt that he really could do valuable work and advance what he always had most at heart—the raising of the standard of obstetric care throughout Australia. So he went, and at irregular intervals I would receive delightful letters telling of his travels and experiences. Letters on the whole "well worth reading—even better worth suppressing"—the more interesting to me as I knew many of his victims personally. His report made obstetrical history in Australia.

From this the next step was obviously the chair and his final departure from Brisbane. He still wrote his vitriolic letters, and I gathered that he was still fighting lions in his path. Whenever I visited Melbourne the hours spent with him had always the effect of a mental shower-bath, and to the note of combat came added a new one—his intense interest in the students who passed through his hands and his pride in their achievements.

The last visits were saddened by the approaching tragedy; he vigorously refused to be sorry for himself, there was still the cheerful welcome, the joy of battle (curtailed though it was by his physical powers), and his plans for the future—plans not for himself, but, as always, for his "boys". His greatest satisfaction was the plan for the M.C.O.G. examination in Australia for 1947—and no doubt his greatest disappointment was that he did not live to see it.

He was intensely outspoken, and the scientific charlatan was the favourite target of his criticism, but however much he might dislike a man or woman personally, if they would work with him he was always ready to hold out a hand to them in cooperation.

He was comparatively young—he came of long-lived stock—but between his devotion to work and his rugged intellectual honesty he burned himself out.

The letter in which he told me of the discovery of his failing health contained not one word of self-pity—only regret that it would interfere with the work he had planned; and perhaps the nearest he came to complaint was a somewhat bitter comparison between a much cried up politician and the medical men throughout Australia who were steadily doing their duty and dying at their posts with no pomp nor recognition. As he grew weaker and as his devoted wife interfered more and more to buffer the blows of his fate, he made of his weakness a joke and turned to discuss some other matter of greater interest and importance to his visitor.

He took the advice of another Edinburgh student, a man who wrote from his own bitter experience:

> It is better to live and be done with it, than to die daily in the sick-room. By all means begin your folio; even if the doctor does not give you a year, even if he hesitates about a month, make one brave push and see what can be accomplished in a week. It is not only in finished undertakings that we ought to honour useful labour. A spirit goes out of the man who means execution, which outlives the most untimely ending. All who have meant good work with their whole hearts have done good work, although they may die before they have the time to sign it. Every heart that has beat strong and cheerfully has left a hopeful impulse behind it in the world, and bettered the tradition of mankind. And even if death catch people, like an open pitfall, and in mid-career, laying out vast projects, and planning monstrous foundations, flushed with hope, and their mouths full of boastful language, they should be at once tripped up and silenced: Is there not something brave and spirited in such a termination? And does not life go down with a better grace, foaming in full body over a precipice, than miserably straggling to an end in sandy deltas?

CEDRIC VICTOR BOWKER.

DR. CEDRIC VICTOR BOWKER, whose death was announced recently in these pages, was one of the sons of the late Dr. Robert Bowker, who practised many years ago at Newcastle and Sydney. Cedric and his elder brother, Robert Steer Bowker, were associated with Sydney Hospital, and will be remembered by those who graduated early in this century as among those who set a standard in surgery at that institution. We are indebted to Dr. R. I. Furber for the following tribute to Cedric Victor Bowker.

Cedric Bowker died recently after a long and trying illness, which he bore with all the calm and resignation that one would have expected of him.

Graduating in 1900, he was a junior resident, resident pathologist and superintendent at the Sydney Hospital, and in 1903 went on the honorary staff as junior gynæcologist till 1919, when he became the senior gynæcologist, and after this remained as consultant gynæcologist till his death. For thirty years he held similar honorary positions at the Royal Hospital for Women.

He began his career as a general practitioner, and this gave him the breadth of knowledge of medicine and the

appreciation of the difficulties under which general practitioners labour, as well as an understanding of humanity so essential in the make-up of a specialist.

He had the inestimable advantage of training under that great gynæcologist and worker for the profession, Ralph Worrall. Taking every advantage of this, he became in his turn a most accurate diagnostician and skilful surgeon, wise with the judgement that comes from thought and experience. He always set a great example for his juniors to emulate, and he was always there for them to turn to in times of trouble. A great teacher, he commanded the respect and affection of us all, staff colleagues, resident and nursing staffs, and above all his many, many patients.

A first-rate tennis player and cricketer and an excellent golfer, his main delight was to spend such leisure hours as came his way in the surf at Maroubra.

The character and soul of a hospital are moulded by such men as Cedric Bowker, and the Sydney Hospital and the Royal Hospital for Women were fortunate indeed to have had his life's service to carry on the torch.

NORMAN JAMES PARKER.

We regret to announce the death of Dr. Norman James Parker, which occurred on August 2, 1946, at Sandringham, Victoria.

JACK HALLING CRAMSIE.

We regret to announce the death of Dr. Jack Halling Cramsie, which occurred on August 23, 1946, at Sydney.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Benedek, Stephen, Provisional Registration, 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Grant, Alan, Provisional Registration, 1946 (Univ. Sydney), Eastern Suburbs Hospital, Waverley.

Bennett, Winston Noel Chevalier Gerard, M.B., 1942 (Univ. Sydney), 3, Gundimaine Avenue, Neutral Bay.

Blashki, Loris Phillip, M.B., B.S., 1941 (Univ. Sydney), c.o. 56, Birriga Road, Bellevue Hill.

Kilgour, Donald William, M.B., B.S., 1944 (Univ. Sydney), Catherine Hill Bay.

Books Received.

"A Treatise on Hygiene and Public Health, with Special Reference to the Tropics", by Birendra Nath Ghosh, M.B.E., F.R.F.P. and S. (Glasgow), F.R.S. (Edinburgh); Eleventh Edition; 1945. Calcutta: Scientific Publishing Company; Sydney: Angus and Robertson Limited. 8½" × 5½", pp. 724, with many illustrations. Price: 21s.

"An Atlas of the Commoner Skin Diseases, with 139 Plates Reproduced by Direct Colour Photography from the Living Subject", by Henry C. G. Semon, M.A., D.M. (Oxon.), F.R.C.P. (London), photography under the direction of Arnold Moritz, B.A., M.B., B.C. (Cantab.); Third Edition; 1946. Bristol: John Wright and Sons Limited. London: Simpkin Marshall (1941) Limited. 9¾" × 7¼", pp. 352, with many illustrations. Price: 50s.

"Squint and Convergence: A Study in Di-Ophthalmology", by N. A. Stutterheim, M.D. (Rand); 1946. London: H. K. Lewis and Company Limited. 9½" × 7½", pp. 104, with illustrations. Price: 15s.

Medical Appointments.

Dr. F. C. Schwarz has been appointed government medical officer at Wentworth, New South Wales.

Dr. S. R. Burston has been appointed honorary consulting physician to the Royal Adelaide Hospital, Adelaide.

Dr. R. W. Klein has been appointed surgical registrar of the Royal Adelaide Hospital, Adelaide.

Dr. H. F. Hustler and Dr. G. H. McQueen have been appointed officers of health, under the provisions of the *Health Act*, 1935-1943, of South Australia.

Dr. A. P. Davis has been appointed visiting medical officer to the "Sunset" Old Men's Home, Dalkeith, Western Australia.

Diary for the Month.

SEPT. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 10.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
SEPT. 13.—Queensland Branch B.M.A.: Council Meeting.
SEPT. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 18.—Western Australian Branch, B.M.A.: General Meeting.
SEPT. 19.—Victorian Branch, B.M.A.: Executive Meeting.
SEPT. 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 19.—South Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

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